A STUDY ON RISK FACTORS AND ENVIRONMENTAL SOURCES FOR ELEVATED BLOOD LEAD LEVELS AMONG PRE-SCHOOL CHILDREN LIVING IN SLUMS OF VELLORE, SOUTH INDIA

A dissertation submitted in partial fulfillment of the requirements for the MD branch XV (Community Medicine) course as required by the Tamil Nadu Dr. M.G.R Medical University, Chennai for the examination to be held in April, 2015.

CERTIFICATE

This is to certify that "**A study on risk factors and environmental sources for elevated Blood lead levels among pre-school children living in slums of Vellore, south India"** is a bonafide work of Dr. Rohan Michael Ramesh in partial fulfillment of the requirements for the M.D Community Medicine, branch XV examination of the Tamil Nadu Dr. M.G.R university to be held in April 2015.

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LIST OF ABBREVIATIONS

BC	Before Christ
BLL	Blood Lead Level
CDC	Centre for Disease Control
DALYs	Disability-Adjusted Life Years.
EBF	Exclusive Breast Feeding
EBLL	Elevated Blood Lead Level
EDTA	Ethylenediaminetetraacetic acid
FAAS	Flame Absorption Atomic Spectrometry
GOI	Government Of India
IQ	Intelligence Quotient
IRB	Institutional Review Board
LCECU	Low Cost Effective Care Unit
MalED	Malnutrition & enteric diseases
MNCV	Maximal motor neuron nerve conduction
NFHS	National Family Health Survey
Pb	Lead
PPM	Parts Per Million
SES	Socio Economic Status
USA	United States of America
WHO	World Health Organization

ABSTRACT

Title: A study on risk factors and environmental sources for elevated blood lead levels among pre-school children living in slums of Vellore, South India.

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Objectives:

1. To study the socio-demographic risk factors associated with elevated Blood Lead Levels among pre-school children.

2. To assess the lead levels in commonly used items in the child's immediate environment.

3. To study the association between environmental sources and elevated Blood lead levels among pre-school children.

Methods:

A community based case-control study was done on 153 participants chosen from the primary study (MalED study). Children from the Mal-ED cohort who have completed 24 months and whose venous blood has been tested for lead levels at 24 months were eligible to participate in this study Children with blood lead levels >/=10mcg/dl at 24 months were assigned as cases and children with blood lead levels <10mcg at 24 months were controls. A masked list of the cases and controls were given to the principle investigator to visit the houses of the participants and administer the questionnaire and collect environmental samples after acquiring their informed consent. The environmental samples were processed and analyzed in Vellore Institute Of Technology. The following variables were studied for each participant:

- Socio demographic factors
- Risk factors: parental occupation, characteristics of the dwelling place, habits, ingestion, breast feeding, cosmetic use and daily activities of the child.
- Environmental samples for lead levels in the child immediate surroundings: wall paint, door paint, house dust, drinking water and cosmetics were tested for lead levels.

Results:

Among the 153 participants, the following are the results:

- Blood lead levels (Primary study)
    - 46.8% of the participants had BLLs >= 10 mcg/dL.
    - 98% of the participants had BLLs >=5 mcg/dL.
- Risk factors for elevated blood lead levels
    - Univariate analysis:
        i. House being painted at least once in the last 5 years.
        ii. Age of the house >=10 years.
        iii. Use of at least 1 cosmetic item (bordering on significance).
    - Multivariate analysis:
        i. House being painted at least once in the last 5 years. [Adjusted OR 7.05 (95%CI 1.84-26.99)]
        ii. Age of the house >=10 years. [Adjusted OR 6.53 (95%CI 2.43-17.58)]
        iii. Habit score of >= 5 habits per child. [Adjusted OR 2.93 (95%CI 1.03-8.37]
        iv. Drinking water stored in plastic containers. [Adjusted OR 8.06 (95%CI 1.04-62.27)]
- Environmental lead levels
    - 91.4% of the drinking water samples had Lead levels more than the CDC recommendation for lead in drinking water.

Conclusions: In conclusion elevated blood lead levels are a major public health problem among preschool children living in urban slums of Vellore. The above mention risk factors portray the various aspects of lead poisoning in pre school children that need to be addressed.

Keywords: Blood Lead Levels, Risk Factors, Environmental Sources, Urban Slums, Preschool Children, Drinking Water Lead Levels.

1. Introduction

Lead is one of the most common heavy elements, accounting for 13 mg/kg of the Earth's crust. It is a soft metal with a melting point of 327°C (1) belonging to group IV A (14) of the periodic table with an atomic number 82 and relative atomic mass 207.2. Pure lead is a silvery white metal that oxidizes and turns blue- grey when exposed to air. The property of lead being soft, dense (11.3 g/cm^3), malleable and readily fusible allows it to be extensively used in a wide variety of applications. Alloying it with small amounts of arsenic, copper, antimony or other metals hardens lead. The use of lead, and the process of extracting lead from ore dates back to ancient times; the earliest known example of metallic lead is a metal statue recovered from the Temple of Abydus in Upper Egypt, considered to date from 4000 BC. Lead is usually obtained from sulphide ores, often in combination with other elements such as zinc, copper and silver. Lead exists in three oxidation states Pb(0) elemental form, Pb(II) and Pb(IV) and has three chemicals forms, viz., metallic lead, inorganic lead compounds and organic lead compounds.(2) But as more and more lead was being used for various applications, more people also started getting exposed to higher concentrations of lead. As time passed by, the effects of lead on the human body slowly surfaced and people began to realize that the toll of lead on the human body was not comparable with its industrial benefits. During the past century the adverse effects of lead on children have been of concern to many, especially since many of its effects are irreversible and incurable. And even as the world undergoes a metamorphosis in development and technology, one thing is for certain –"prevention is the best way to deal with lead poisoning".

2. Hypotheses

2.1. Elevated Blood lead levels among pre-school children are associated with the socio-demographic characteristics of the family, parental occupational characteristics and child behaviours.

2.2. The living environment of the children constitutes an important source of exposure to lead.

3. Objectives

3.1. To study the socio-demographic risk factors associated with elevated Blood Lead Levels among pre-school children who have completed 24 months of age living in slums of Vellore.

3.2. To assess the lead levels in commonly used items in the child’s immediate environment.

3.3. To study the association between environmental sources and elevated Blood lead levels among pre-school children who have completed 24 months of age living in slums of Vellore city.

4. Justification

Lead poisoning has been scarring human health for centuries now. Since the beginning of the 20^{th} century, childhood lead poisoning has been recognized as an important clinical entity. Children are affected with a wide spectrum of permanent conditions ranging from mental retardation and behavioural disruption at higher levels of exposures to cognition Impairment, dyslexia, decreased attention span, alteration of behaviour, attention deficit disorder, renal impairment and damage to reproductive organs even at lower levels of exposure to name a few.(3) Elevated Blood lead levels are more commonly associated with children in poor living conditions and low socio economic sections of society like slum dwellers. (4) As children form one the most vulnerable sections of society, safe guarding them against the ill effects of lead becomes a prime public health responsibility.

In India, many studies have been conducted to estimate the burden of lead poisoning and its risk factors, which have shown that even after the decline in leaded petrol, lead poisoning continues to be a public health problem. Though there are many cross sectional studies conducted, there are very few community based risk factor studies assessing environmental sources for elevated BLLs in India.

This study is a community based case control study, which aims to study the socio-demographic risk factors for elevated blood lead levels and assess important environmental sources of lead among pre-school children living in urban slums of Vellore city in the state of Tamil Nadu, South India.

5. Literature review

5.1. History

Lead has been identified as a toxin as early as 2000 BC. The Romans were one of the first civilizations to get affected due to the extensive use of lead in earthenware, water pipes and wine storing containers. They also used to drink wine sweetened with a leaded syrup called '*sapa*'.(3, 5)

Lead poisoning due to occupational exposure has been reported as early as 370 BC.(3) In 1767, Benjamin Franklin one of the founding fathers of the United states and a great inventor was given a list of patients admitted in La Charite hospital in Paris with symptoms suggestive of lead poisoning, all the patients were exposed to lead in their respective occupations.(3, 6) The signs and symptoms of lead poisoning were thoroughly documented by the French physician Tanqueral des Planches in 1839 from in his study of 1213 patients admitted to the La Charite hospital-Paris.(7)

In the 19^{th} and early 20^{th} centuries lead poisoning was quite commonly noted among industrial workers involved in painting, plumbing, smelting and printing.(3, 8) But following the deaths of several employees in the lead industry, in 1883 a parliamentary enquiry was initiated to look into the working conditions of these factories in London.(3) This resulted in the Factory and Workshop Act in 1883 where lead factories in the United Kingdom had to conform to minimum standards of ventilation and protective clothing.(8)

In Australia, lead poisoning was recognized as a pediatric health problem more than a century ago, when 10 cases were reported in Queensland in 1892 due to lead based paint on verandas of the children's homes.(3, 9)

5.2. Global scenario

In developing countries, almost all children under 2 years and > 80% of those between 3 to 5 years are suspected to have BLLs more than 10μg/dl. It has been estimated that more than 18 million children in these countries suffer from permanent brain damage.(10, 11) In Karachi, a study showed that about 80% of children aged 36 to 60 months had elevated BLLs>10μg/dl with an overall mean of 15.5μg/dl.(12) In a study among Saudi Arabian school children, 6% had BLLs >10μg/dl.(13) A study in Jakarta, Indonesia showed 35% of the school going children between 6 to 12 years had BLLs >=10μg/dl.(14) Twenty one percent of Pilipino children between the age groups of 6 months to 5 years were found to have elevated BLLs.(15) Nearly 10 % of children under 6 years of age in the USA had blood lead levels more than 10 mcg/dL in 1999.(16) Nearly 10 % of children under 15 years of age in the African region, 34% in the Mediterranean region and 24 % in the south east Asian region have elevated blood lead levels.(17) The first country to reduce lead content in petrol was Japan in 1970, following widespread contamination in Tokyo.(11) In 1994, the United Nations commissions called on Governments worldwide to switch to unleaded petrol.(10) But by 1996, only 14 countries had completely phased out leaded petrol, out of the which very few were African and Asian countries.(11,18) Unleaded petrol contains ethanol or manganese as a replacement for lead as an anti knock in petrol.(11) After the United States started implementing measures to control lead pollution in the 1970s, BLLs began to decrease by more than 80%.(19) The largest environmental lead exposure source in the world is leaded petrol.(11,20) Today in the United States, lead based paints are an important source of lead poisoning, but in Mexico lead glaze coated ceramics used for storage and preparation of food are identified as important lead sources.(21) The success in elimination of lead from petrol worldwide was short lived, as globally people started realizing that though lead poisoning was on the decline there were other sources of lead, which have to be dealt with. Disability-adjusted life years (DALYs) are “the sum of years of life lost due to death and to disability due to a particular disease or condition.”(3) Each of these conditions is associated with a defined severity weight and DALYs are the metric used by World Health organization to assess the global burden of disease worldwide. The total burden of disease attributable to lead, amounts to nearly 9 million DALYs, representing 0.6% of global burden of disease. In 2004, it was estimated that 16% of children worldwide had BLLs greater than 10μg/dl and 90% of these children lived in low-income regions.(3)

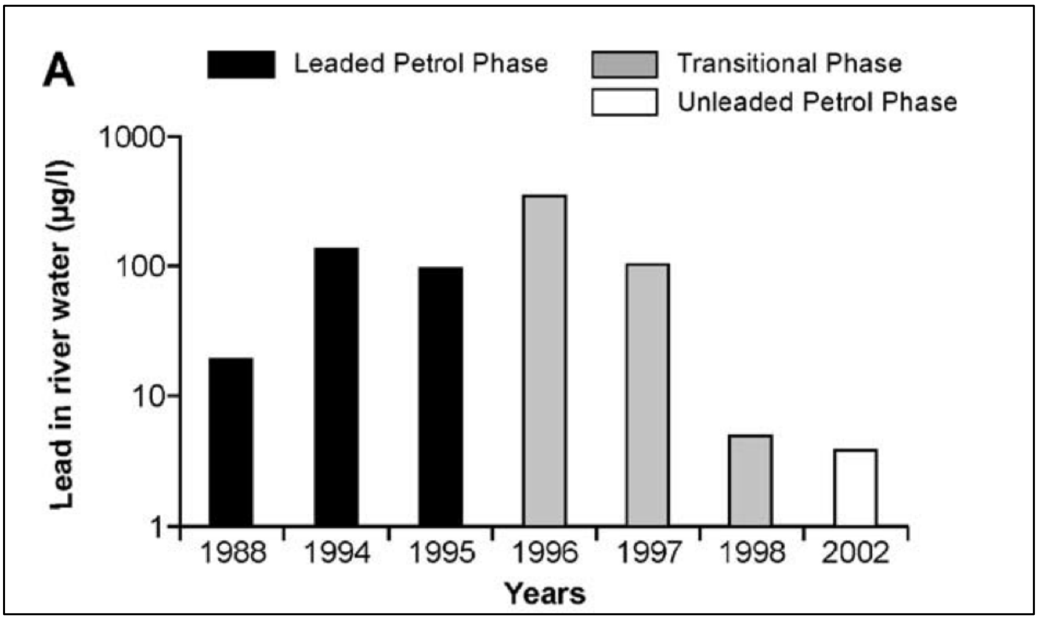
TABLE 1: Elevated BLLs from several studies worldwide.

Sl. No.	% Children with elevated BLLs ($\geq$10 μg/dL)	Target population	Year	Setting	Source
1	7.6 -10.5%	Children under 6 yrs.	1996 - 1998	19 US states	NHANES report; 1999(16)
2	43%	100 children aged 6 to 12 yrs.	2007 - 2008	Cairo, Egypt	Mostafa et al.(22)
3	12%	Children < 15 yrs.	2004	LMIC-African Region	GHOD, WHO(17)
4	34%	Children < 15 yrs.	2004	LMIC- Eastern Mediterranean Region	GHOD, WHO(17)
5	2%	Children < 15 yrs.	2004	LMIC-European Region	GHOD, WHO(17)
6	24%	Children < 15 yrs.	2004	LMIC- South East Asia Region	GHOD, WHO(17)
7	21%	2861 children aged 6 mths.to 5 yrs.	2003- 2004	Rural Philippines	Riddel et al.(15)

5.3. Indian scenario

Unlike most developed countries, lead poisoning continues to be a public health concern in developing countries like India, where laws and legislation regarding lead use were implemented rather late as compared to the rest of the world.(3) In the latter half of the 20th century, urban environments across the country experienced drastic changes as small towns grew into industrial cities, cities to metropolises and metropolises to megalopolises. This meant that much more people were exposed to the lead via leaded petrol.(11) To address this issue, the Ministry of Environment and Forest and the Ministry of Petroleum and Natural Gas, Government of India introduced initiatives to reduce leaded petrol in a phased manner in the metropolitan cities for the first time in 1995 and finally completely phase out leaded petrol throughout the country by April 2001.(11,23) The figure below shows phases of introducing unleaded petrol in India.

FIGURE 1: Phases of elimination of leaded petrol in India(11)



Source: Lead decline in the Indian environment resulting from the petrol-lead phase-out programme.

Various studies have monitored lead levels in children, water and atmosphere by bio monitoring of trees and other methods in India. Even the river Ganges showed reduction of lead concentrations from 1988 to 2001. (11) There was a decrease in mean air lead concentration by 87.5% in Lucknow between 1994 to 2002. (24) Mean blood lead levels of children from Mumbai, Chennai, Bangalore, Amritsar and Lucknow urban centers decreases from 18.1 mcg/dl in the leaded petrol phase to 12.1 mcg/dl/dl in the unleaded petrol phase.(11)

As leaded fuel began to slowly move out of the picture, BLLs slowly began to decrease as the emissions from the vehicles that used leaded petrol dwindled in numbers. In 1997, a study from Delhi revealed that among school children aged 6 to 10 years, 55.6% of the children hailing from low ambient air lead levels and 72.3% of the children hailing from high ambient air lead levels had BLLs> 10 μg/dl.(25) Another study during the same period conducted in Delhi, showed 54.1% of the participants who were aged <12 years had BLLs more than 10 μg/dl and 18.6% participants <12 years had BLLs> 20 μg/dl. (25, 26) In 2006, 8 years after phasing out of leaded petrol, a study among school children showed that 12% of the 300 school children had elevated BLLs.(25) In other similar studies 37% of children in Mumbai and > 50% of Children in Chennai had BLLs>10μg/dl in 2003 and 2006,(27-29) suggesting that children are still exposed to lead in spite of eliminating a major source, which was leaded petrol.

In a cross sectional study conducted in a south Indian slum in Vellore almost half of the children had BLLs exceeding the CDC cut off of 10 μg/dl at 15 months of age. (4) In another study conducted in an urban slum in South

Delhi, 18.4% of the children between 4 to 6 years of age had elevated BLLs, these results are similar to the BLLs observed in developed countries in the late 1970s.(30, 31) This suggests that in India, lead poisoning among children is an important public health problem and there is a need to focus on multiple sources of lead exposure like drinking water, dietary intake, lead glazed ceramics, cosmetics, lead based paints, lead based batteries, and lead soldered food cans.(32)

Some other factors which were significantly associated with BLLs in the National Family Health Survey 3 (NFHS-3) by the Government of India (GOI), included age, standard of living, height /weight percentile and total number of children born to that mother.(19) Many studies have also showed poor socio economic status to be associated with elevated BLLs.(30, 33) Lead poisoning in India will continue to be a challenge as cities rapidly industrialize and swell to its limits, but local, national and international coordination will help ensure a lead free India for children in the future.(11)

5.4. Uses of lead

Lead has been extensively used in a wide variety of industries and is commonly used in producing lead acid batteries, alloys, solder, cable sheathing, pigments, rust inhibitors, ceramic glazes and plastic stabilizers. Lead was one of the key components of petrol used as a antiknock compound but has gradually been phased out in most countries. Lead compounds have extensively been used in plumbing fittings, pipes and as soldering material in water distribution systems contributing to water contamination. (1) Majority of the lead produced is from China, Australia, the United States and Peru. But the world's consumption of lead is significantly larger for it includes recycled lead. In 2010, 9.6 million tons of lead was produced, of which 4.1 million tons came from mining. At this rate at which lead is used, the supply of lead is estimated to run out in 42 years. Predominantly (71%) lead is used in vehicular batteries and electricity backup systems, followed by pigment (21%), ammunition (6%) and cable sheathing (3%).(34)

5.5. Lead poisoning sources

Lead has unique characteristics like its high density, softness, ductility, malleability, poor electrical conductivity, high corrosion resistance, and its ability to react with organic chemicals makes it a popular choice for multiple applications. Due to the its extensive use, lead is dispersed in air, dissolves in water and ultimately finds its way into the human blood stream. Lead is found in the solar atmosphere and metallic lead rarely occurs in nature. Lead is also found in ore with zinc, silver and mostly in copper, the main lead mineral is galena, which contains 86.6% lead by weight.(3)

5.5.1 **Air:**

Lead concentration in air depends on various factors like proximity to roads and point sources. High concentrations of lead in air were noted when there were battery plants and lead smelters in the vicinity. Vehicular sources of lead have slowly and steadily been declining after the use of lead in petrol has been phased out in many countries. Open burning of waste especially in landfills to reduce the volume of waste are one of the main sources which introduces lead into the environment. (3) Often waste which are thought to have metals in them are brought home by scavengers to extract metals especially from e-waste or waste from electronic items. Such waste may also be used as cheap combustible material used inside homes to cook or heat. Incinerators, cement kilns and crematoriums may also emit large amounts of lead into the air that may pollute many communities settled even kilometers away from the sources. If the concentration of lead in air is 0.2 $\mu g/m^3$, the intake of lead in air would range from 0.5 μg/day for an infant to 4 μg/day for an adult. (1) According to the WHO air quality guidelines, the annual average lead level in air should not exceed 0.5μg m^3.(35) In one Indian study in central India the mean air lead levels in Raipur, Bhilai, Kaudikasa, Mandla and Korba were95 ±18 μg m^3 which was way above the recommended WHO level. (36)

5.5.2 **Water:**

A major source of lead is from contamination of drinking water, which mainly occurs from plumbing systems at homes via pipes, taps and solders fittings containing lead. Polyvinyl Chloride (PVC) pipes also leach lead resulting in high lead levels in drinking water. Chloride, dissolved oxygen, pH, temperature, softness and standing time of the water affect the lead concentrations in water. Copper piping can also increase lead concentrations to the extent that it can intoxicate children. (1) Increasing the pH of the water from 8 to 9 and adding lime can control corrosion of pipes. (1)

The mean lead level in the USA in drinking water was 2.8 μg/l in 1990. In the UK there was no detectable lead in two thirds of the households with only 10 percent of the homes with lead levels exceeding 50 μg/l. (1) In a South Indian study, 85.5% of the houses were supplied with drinking water through local municipal public taps, and having a piped water supply was significantly associated with elevated BLLs among children.(4) If the concentration of lead in water is 5 μg/l, the intake of lead from water would range from 3.8 μg/day for an infant to 10 μg/day for an adult. (1) The WHO standard for lead in drinking water is 10μg/l (0.01ppm).(37)

5.5.3 **Food:**

Food contains small but significant amounts of lead.(1) The main source is through contaminated water when used for cooking or using cooking utensils containing lead like lead enamel ceramic pottery(38) and lead soldered cans. In India, brass vessels are also coated with lead to prevent corrosion and to enhance the taste of the dish cooked in it and it is not uncommon to find brass vessels in Indian households, which are used for cooking and storing water. Adults nearly absorb as much as 10% of lead in food. Up to 50% of lead can be absorbed by children as compared to 10% in adults, due to increased gastrointestinal absorption.(1) Increased hand to mouth activities also makes children more susceptible to lead poisoning. The use of any lead based product in the manufacture of production of alcohol tends to raise the lead content due to the acidic nature of alcohol. Tobacco smoking also increases lead intake.(3) The soil concentration of lead is highest around mines and smelters, which affects the amount of lead in food plants like cereals. Lead in milk and formula feeds continue to be an important source of lead in food for infants.(3)

5.5.4 **Paints:**

Paints continue to be an important source of lead poisoning in many developing countries. Lead is added to paints to enhance color, increase durability, improve drying and to make it corrosion resistant.(2) Lead oxide, lead carbonate and lead chromates are commonly used as pigments in paints. Pigments are solid portions of the paint, which are added to enhance color and durability of the paint. The most common pigment being lead carbonate and paints containing lead pigments continue to be a source of lead poisoning long after the paint is applied. There are still around 38 million housing units in the United States that have lead in their paints even though lead was banned in 1978.(39) As older homes undergo renovation or lead based paints start to deteriorate lead containing dust is generated and dispersed into the immediate environment. The United States in 1978 banned paints containing more than 0.06% (600 ppm) lead on toys, furniture, interior and external surfaces of houses, buildings and structures used by general public.(3) In 2009, the United States banned any product intended for use by any child <12 years of age, which contained more than 300-ppm total lead per weight. Paint in furniture, toys and other products also should not contain more than 0.009% or 90 ppm by weight. Children may directly or indirectly come in contact with leaded paint in the paint itself or the interior dust and exterior soil or dust. Children especially are more susceptible to lead poisoning by this source due to increased hand to mouth activity. As paint ages it peels or chips off. The sweet taste of lead and the bright colored paints attract children to taste and eat the peeling paint out of curiosity.(40) Even if they don’t intentionally eat the material, the dust can get on their hands and into their food.

In India due to lack of regulations and stringent laws, lead continues to be added in paints causing lead poisoning in children. Studies show that 84% of enamel paints being sold in India for residential purpose contained >600 ppm of lead per weight. Indian studies show that if the lead in paints exceeds 10000 ppm it could prove to be hazardous.(2) The laws in the USA permit lead levels <0.06% but most Indian paints contain up to 50% lead salts. Indian standards allow up to 1000 ppm of lead in paints. Yellow, brown and orange paints usually contain high levels of lead. These being bright colors are extensively used in children play equipment. Even though yellow contains high levels of lead and most school buses are painted with it.(2,40)

The use of lead based paints for homes have been banned since the 1970s in the USA and therefore older homes and building often retain remnants of older paints. In a study conducted in the USA, children living in older homes had higher geometric mean concentration of BLLs as compared to newer ones and from 1900 to 1980, the older the house, the higher the mean blood lead level of resident children and the greater the proportion of resident children with elevated blood lead levels.(41)

5.5.5 **Parental exposure:**

Another important source contributing to lead poisoning in children is through parental exposure. Parents who work in industries that use lead may bring back lead to their homes through their clothing, shoes and vehicles.(3) Many cases of lead poisoning of the children and spouses of those who work in lead rich environments have been documented.(42,43) Elevated BLLs have also been noticed in many instances where children have worked as child laborers, scavengers, street vendors, car repairers and ship dismantlers. The list of occupations with significant exposure to lead is outlined below:

High-risk occupations:

- Plumbers/ pipe fitters
- Lead miners
- Lead smelters and refiners
- Auto repairers
- Glass manufacturers
- Ship builders
- Printers
- Plastic manufacturers
- Police officers
- Steel welders/ cutters
- Construction workers
- Rubber product manufacturers
- Gas station attendants (past exposure)
- Battery manufacturers
- Battery recyclers
- Bridge reconstruction workers
- Firing range workers
- Painters
- Electrician/electronic work

Other factors like duration of work in years, type of work, change in work clothes and bathing and hygiene have also been shown to be associated with lead poisoning among children.

5.5.6 Prenatal exposure and exposure to lead in human milk:

Lead that was stored in the mothers' skeleton is released into circulation due to the metabolic stress of pregnancy. The maternal lead circulating readily crosses in to the fetal circulation and the concentration of lead becomes identical to that of the mothers.(44) The lead crosses the blood brain barrier and enters the developing brain of the infant, which is susceptible to lead even at low levels of exposure.(45) The lead in an infant's blood is a mixture of one third skeletal lead and two thirds dietary lead.(46) The concentration of lead in human milk is similar to that of plasma lead but much lower than that of whole blood lead, which means there is a little amount transferred to the infant. The water used to make infant formulae and other food may also contain lead, so breastfeeding may be a protective factor if external lead exposure is high. Calcium supplementation has been proven to decrease blood lead concentrations antenataly in some studies done in Mexico, therefore decreasing the transfer of lead through breast milk.(47)

5.5.7 Lead glazed ceramics:

It is not uncommon to find high lead content used in the glaze applied on ceramics and acidic foods, cooking and older cracked pots increase the amount of lead, which leaches from the glaze. These lead glazed pots are preferred in some places as it adds a distinct flavor to the food cooked in it. In countries such as Ecuador, lead salts made from melted batteries are used to glaze ceramics.(3)

5.5.8 Lead in electronic wastes:

With the global increase in computers, cell phones and other electronic items, a large amount of electronic waste is generated as instruments become obsolete and non-functional. A lot of these items nearing the end of its usefulness are shipped to low-income countries for lead, mercury and other metals to be separated from the waste for recycling and reusing.(3) Communities engaged in such work have been found to possess elevated lead levels in the dust and children in this profession also have elevated BLLs.(48,49) In Manila, a survey of 231 scavengers between 6 to 15 years recorded a mean blood lead level of 28.4μg/dl as compared to the mean blood levels of 11μg/dl in 25 school children.(50)

5.5.9 **Other sources:**

Household dust is another important source of lead. Lead levels of house hold dust in Delhi homes is much higher than that in the national data in the USA.(51) Dust from renovation sites and remodeling remedies also contains high concentrations of lead.(52)

In a South Indian study, 16 % of the houses had mud walls and 6% had floor made of sand, mud or clay, which was significantly associated with elevated BLLs.(4) It is a known fact that heavy metals are added to Ayurvedic medicines.(41) Around 80% of the Indian population relies on traditional systems of healthcare like Ayurveda for various ailments like infant colic, teething, colds, diarrhea and other health conditions. But a number of cases of lead poisoning associated with Ayurvedic products have raised health concerns all over the globe and not only in the Indian sub-continent. Reports range from lead encephalopathy, severe developmental delay and congenital paralysis to deafness in preterm infants.(53)

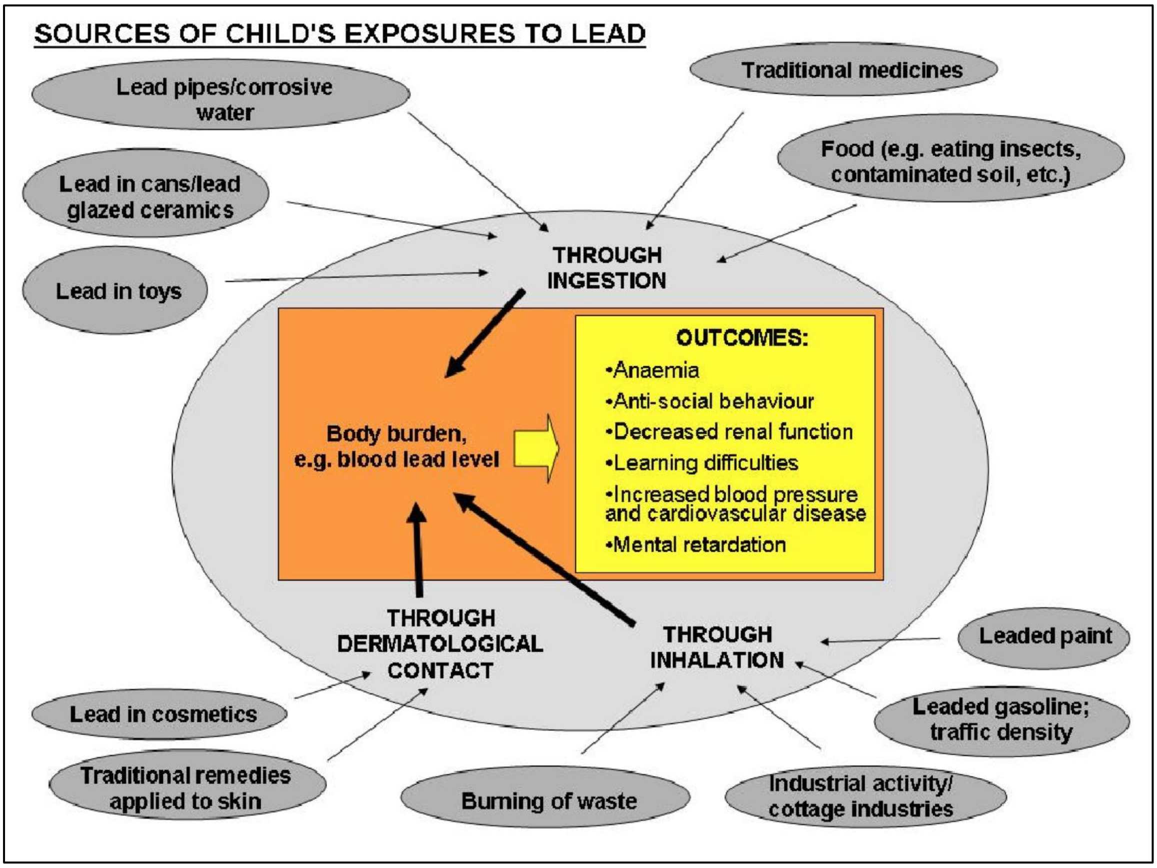
Eye cosmetics like kajal, surma (a powder applied to the eyes) and kohl are used commonly in many Arabic and Asian countries are known to contain lead.(52,54)

Toys are also an important source of lead exposure in children and may be coated with leaded paint or may be made of lead itself. In 2007, Mattel recalled 9,67,000 toys from the market because lead paint standards were violated in these toys in the United States.(32,55) There have also been instances of children swallowing lead coated trinkets or medallions which have ended in death or very high levels of blood lead in blood.(56) Lately there has been an increase in Chinese toys flooding the Indian market. It is of concern to us because many countries have banned the import of these toys due to high lead concentrations detected in them. (4,57) High concentrations of lead in paint (up to 145000 μg/g) used in toys purchased from large stores as well as roadside shops were found in one study.(58)

Open burning of improperly segregated waste and informal recovery of lead from car batteries are important sources of lead in the developing world.(21)

Socio economic factors play an important role in lead exposure to poor families. These families usually live in industrial areas that handle lead to recycle batteries or near smelters. The surrounding industries may also be polluting the area with waste high in lead content or live in old houses with lead based paints.(3)

FIGURE 2: Primary sources and exposure to lead in children(3)



*Source: Childhood lead poisoning -WHO(*www.who.int/ceh/publications/leadguidance.pdf)

5.6 Environmental lead estimation methods:

There are various validated methods to analyze lead in environmental samples like: Chemical test kits- these are portable quick test kits which can be used in the field to determine if there is a presence of lead in a sample however it will not quantize the level of lead. Portable X ray fluorescence spectrometry- this is a portable method used to determine the quantity of lead in solid samples in the field, Flame atomic absorption spectrometry (FAAS) and Graphite furnace atomic absorption spectrometry (GFAAS) are laboratory methods used to determine the quantity of lead in samples which have to be dissolved into a liquid for analysis. Inductively coupled plasma atomic emission spectrometry

(ICP-AES) is also another quantitative laboratory method that is recommended but the only drawback is the cost. (59)

5.7 Metabolism

The most common route of lead poisoning is ingestion followed by inhalation and very rarely via skin. The red blood cells help transport lead from the intestines to different tissues in the body.(1) The lead is bound to the beta, delta and fetal gamma chains in the hemoglobin of the red blood cells. It then appears in the liver, spleen, lungs, kidneys and bone marrow and slowly appears in the bones. Although the half-life of lead in the adult blood is just 36-40 days, in the skeletal pool it lasts for more than 17-27 years.(1) Nearly 90% of the total body lead is found in adult bones unlike 73% in children. The half-life of lead is longer in children and blood lead level is a good indicator of exposure from various sources. Lead can also cross the placental barrier by 12 weeks of gestation and the fetus continues to metabolize throughout development. The BLLs in the umbilical cord is >80% of the maternal blood lead level. Inorganic lead is not absorbed in the body but eliminated in the faeces. The lead that is not metabolized but absorbed is excreted through the kidneys or biliary tract. Children absorb 4-5 times more than an adult. Absorption is even more in children with pica and nutritional deficiencies.(3, 4, 60, 61) Children tend to be more vulnerable to leads toxicity even at low exposure levels causing serious and irreversible neurological effects.(3, 4, 62, 63) Anemic children and children with low levels of calcium and phosphorus are more at risk of absorbing lead than others.(1)

5.8 Effect of lead in humans

5.8.1 Lead toxicity in children

Children particularly are at increased risk of exposure to lead because they are exposed to lead throughout pregnancy. They also drink more water, eat more food and breathe more air per unit body weight.(64) Due to their innate curiosity and hand to mouth behavior and occasionally pica they ingest more lead as compared to adults. They also spend more time in one environment such as the home. Children are also more likely to be nutritionally deficient and lack circumstantial control over their immediate environment that facilitate absorption of lead.(61)

Children are more biologically susceptible to lead than adults due to various reasons lead exposure in early life can re-programme genes which sometimes leads to altered gene expression and later in life, increased risk of disease.(65-67) Early exposure to lead also reduces the capacity to weather any other neurological insults later.(3,61,68) Up to 50% of lead can be absorbed by children as compared to 10% in adults, due to increased gastrointestinal absorption. Childhood exposure can also affect immune system development and lead to immune dysfunction later, though the effect may be latent and may surface only when the immune system may be stressed even if the exposure was long stopped.(3) Lead can interfere with many delicate and complex processes during human brain growth development and differentiation. This damage to the brain is irreversible and untreatable which extends from prenatal life to early childhood.(69,70)

5.8.2 Signs of acute intoxication:

When BLLs in adults reach 100-120 μg/dl and 80-100 μg/dl in children, signs of acute intoxication are evident. Dullness, restlessness, irritability, decreased attention span, headaches, muscle tremors, abdominal cramps, kidney damage, hallucinations, memory loss, and encephalopathy are overt signs of acute lead poisoning in adults. In children intense high dose exposure leads to colic, fatigue, constipation, anemia, poor appetite and even stupor. Very high exposures can present with acute encephalopathy, ataxia, convulsions and even coma. This can lead to deficits, which are permanent and clinically apparent in their neurodevelopmental function.(3,71)

5.8.3 Signs of Chronic intoxication:

When BLLs in adults reach 50-80 μg/dl signs such as sleeplessness, irritability, headaches, joint pain and gastro intestinal symptoms begin to surface.(3)

At BLLs of 40-60 μg/dl with a chronic exposure for about 1-2 years, lower psychometric test scores, mood disturbances, symptoms of peripheral neuropathy were noted especially in populations where the source was through occupational exposure.(3)

Hematological toxicity:

5.8.3a Anemia is the only clinically significant symptom due to the effect of lead in the biosynthesis of haem. This is usually seen at BLLs more than 40 μg/dl in adults and 50μg/dl in children. Lead inhibits haem synthesis and accelerates the destruction of erythrocytes.(1) Iron deficiency and younger age are probable risk factors for developing lead induced anemia and its prevalence and severity correlates well with BLLs.(4,13,72,73) Many studies show that anemia is more common in children below 3 years of age with elevated BLLs.(4,27)

Lead inhibits ferrochelatase, which leads to the accumulation of erythrocyte protoporphyrin. The accumulation of erythrocyte protoporphyrin indicates mitochondrial injury.(1) Changes in growth pattern have been noticed with children less than 42 months and with elevated erythrocyte protoporphyrin levels. There is a rapid gain in weight followed by retardation of growth in these children with BLLs ranging from 5-35 μg/dl.(1)

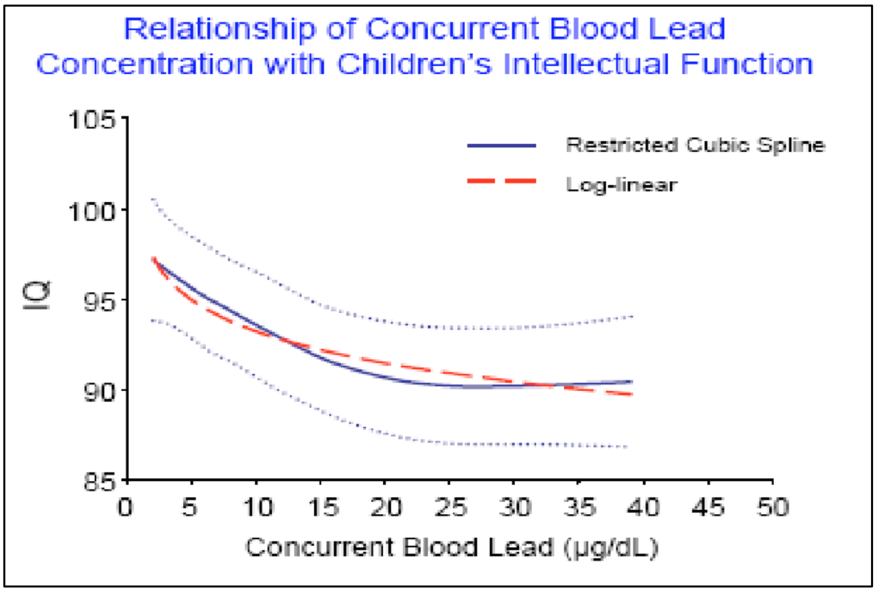
5.8.3b Calcium metabolism is affected by lead directly and indirectly by affecting haem-mediated generation of vitamin D precursor 1,25-dihydroxycholecalciferol. It has been noticed that there is a significant decrease in 1,25-dihydroxycholecalciferol levels in children when the BLLs range from 12-120 μg/dl. The amount of lead in tissue is also increased with persons with calcium deficiency. This is particularly important with regard to pregnant women who are more sensitive to lead exposure due to their calcium deficient states. General intelligence is also affected due to the interactions between calcium and lead.(3)

5.8.3c Neurological toxicity:

The central and peripheral nervous systems are primarily affected by lead toxicity. The motor neurons are the principal target of lead toxicity in the peripheral nervous system, inducing pathological changes in the fibers causing segmental demyelination and axonal degeneration.(3) Lead causes asymptomatic impairment of neurobehavioral function in the central nervous system of children, even at doses which are insufficient to cause clinical encephalopathy. Effects of lead can range from sub encephalopathic neurological and behavioral effects in adults to electrophysiological evidence of effects in the nervous system in children with blood levels below 30 μg/dl. Maximal motor neuron nerve conduction (MNCV) have been significantly been reduced in children aged 5-9 years with BLLs around 20 μg/dl. The auditory nerve may also be affected leading to decreased hearing acuity in children. In children with lead exposure behavior and cognitive development are adversely affected.(4, 62, 63, 73) An association between cognitive functions at a later age with elevated childhood BLLs has also been documented.(4, 74-76)

In a South Indian study, cognitive scores at 15 months of age were negatively correlated with BLLs.(4) A systematic review indicated that doubling of the BLLs was associated with a mean deficit of 1 to 2 points in the full scale IQ.(4, 77) A study of urban children aged 3 to 7 years residing in Chennai, India, found that reduced visual motor abilities was associated with elevated BLLs.(4) Even with low levels of lead exposure an association with intellectual impairment in children was seen.(25, 78) There is also a steep decline in visual and motor skills at higher BLLs.(79) Sub clinical neurobehavioral toxicity is associated with young children whose whole BLLs are as low as 1-3 μg/dl.(80) In the Third National Health and Nutrition Examination Survey (NHANES) conducted in the United States, children between 6 to 16 years of age were found to have an inverse relationship between BLLs and math and reading scores even with blood lead concentrations as low as 5 μg/dl.(81) Currently it is believed that no lead level is safe for infants and young children.(11, 74, 82) Children are particularly at risk of developing problems with their intellectual abilities and behavior at blood levels lower than 40 μg/dl where clinical signs and symptoms are usually not present.

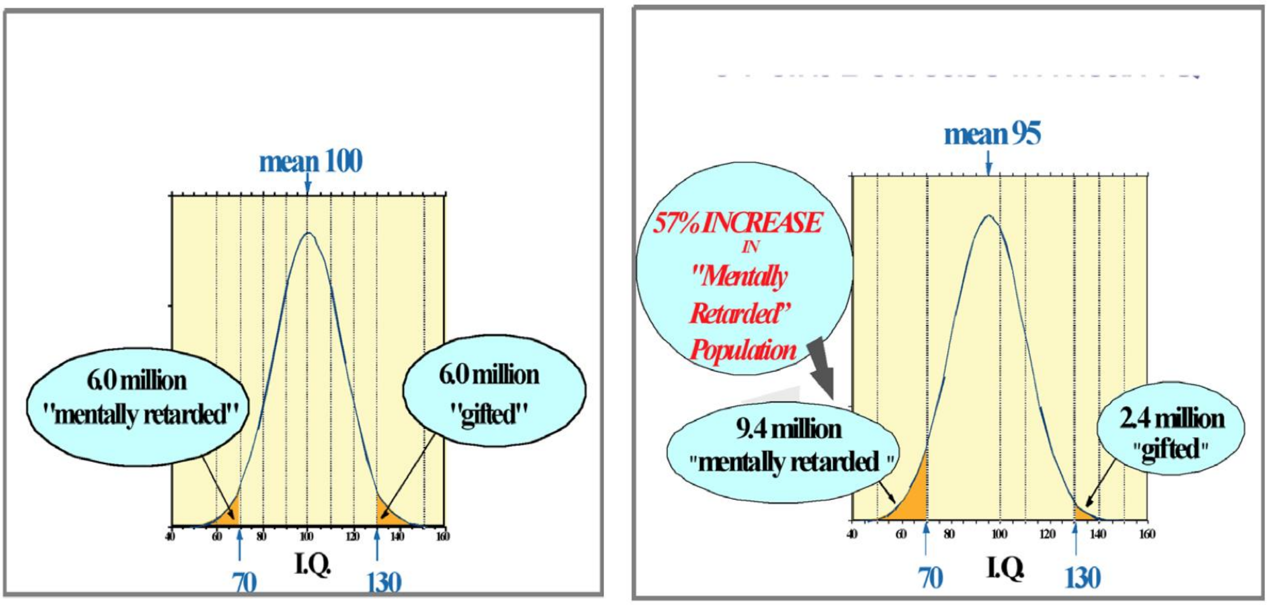
FIGURE 3: Relationship of BLLs and intellectual function in children.(3, 81)



Source: Childhood lead poisoning –WHO (www.who.int/ceh/publications/leadguidance.pdf)

When there is a decrease in mean IQ in populations exposed to large amount of lead, the number of children with diminished intelligence and mental retardation substantially increase and the number of children with superior intelligence decrease.(3, 81) The consequences of this would mean that the number of children who perform poorly at school increase, which would indirectly increase school dropouts and this special group may also require special classes or remedial programmes. When this cohort become adult, their contribution to the society would also drastically decrease and there would be a considerable reduction in the countries future leadership. It has been postulated that this could widen the socioeconomic attainment between countries.(83)

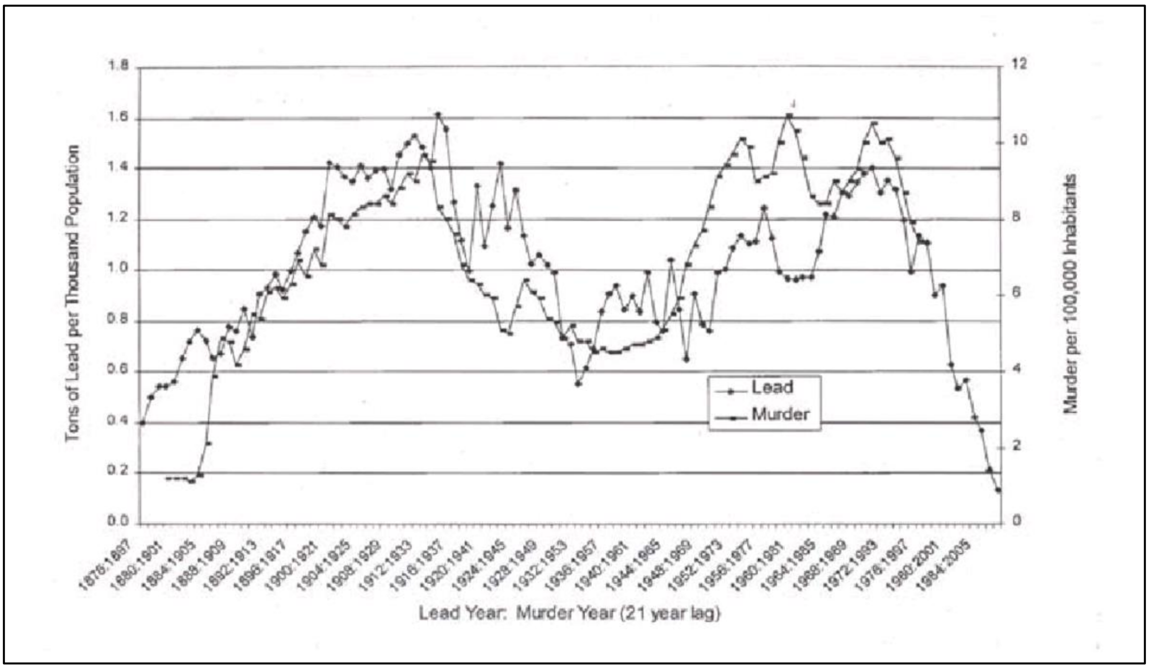
FIGURE NO. 4: Losses associated with a five-point drop in IQ in 100 million people.(3, 84)



Source: Childhood lead poisoning –WHO (www.who.int/ceh/publications/leadguidance.pdf)

Early exposures have also been associated with increased hyperactivity, inattentiveness, failure to graduate from high school, conduct disorders and juvenile delinquency, drug use and incarceration.(3) It was also observed in the United States that murder rates sharply fell after a lag period of 20 years after the removal of leaded gasoline.(85)

FIGURE 5: Correlation between mean BLLs and murder rate, United States, 1878-2006.(3, 85)



Source: Childhood lead poisoning -WHO (www.who.int/ceh/publications/leadguidance.pdf)

5.8.3d Lead and renal toxicity:

In adults and children chronic nephropathy has been noted with BLLs even lower than 40 μg/dl. Renal damage with acute proximal tubular dysfunction is characterized by lead protein complex inclusion bodies, which is associated with increased risk of nephropathy and renal failure at concentrations between 40-80 μg/dl. Diabetics and hypertensives are at increased risk of renal dysfunction even at lower exposures.(3)

5.8.3c Lead and cardiovascular disease:

In the early 20th century, occupationally exposed workers who were exposed to long-term high dose exposure to lead had increased incidence of hypertension and stroke. Studies in the United States show that even at lower levels of exposure to lead, the general population tends to have elevated blood pressure, even with no occupational exposure. BLLs greater than 37 μg/dl is associated with hypertension.(3) Increased exposure to lead has been associated with cardiovascular events which adds to the total economic costs of lead poisoning.(86)

5.8.3f Lead and reproductive function:

Reproductive function in men may be affected with gonadal dysfunction and decreased sperm counts with BLLs ranging from 40-50 μg/dl. Females may also experience sexual dysfunction when occupationally exposed to lead. Pregnant women who are exposed to lead and with a blood lead level of above 14 μg/dl are 4 times more at risk of preterm delivery than those with less than 8 μg/dl.(86)

Many studies have also shown that lead acts an abortificant in pregnant women. (11, 87-89)Minor malformations like angiomas, syndactylism and hydrocele are associated with 10% of babies with increased BLLs. The risk nearly doubles with BLLs ranging from 7-10 μg/dl.

5.8.3g Lead and carcinogenicity:

Chromatid and chromosomal aberrations, breaks and gaps were seen in cytogenetic studies with BLLs more than 40 μg/dl.

Smelters with BLLs more than 80 μg/dl and battery factory workers with BLLs more than 63 μg/dl were more at a risk of dying from a cancer of the respiratory, digestive system or urinary tract. Inorganic forms of lead are more carcinogenic to humans.

5.9 Elimination of lead from the human body:

5.9.1 **Kidneys:** The excretion of lead in urine is through glomerular filtration followed by partial tubular absorption with decrease in excretion during the night. This is one of the main modes of excretion.(34)

5.9.2 **Gastro intestinal tract:** Lead is also excreted through bile, pancreatic juice into faeces.

5.9.3 **Other routes:** Lead is also excreted in small amounts through sweat, saliva, nail, hair, semen and milk.

5.10 Clinical indicators for blood lead testing:

Blood lead testing is not indicated for all children, except if there is a suspected presence of exposure of a risk factor, physical signs and symptoms of lead poisoning are present, any member of the household with a known exposure to lead and acutely ill children with severe colic, seizure or coma.(3)

5.11 Treatment of lead poisoning:

Treatment with a chelating agent usually Ethylenediaminetetraacetic acid (EDTA) is indicated in infants with lead induced encephalopathy for 5 days. Intravenous British Anti Lewisite is usually started before and throughout chelation to prevent transfer of chelating lead to the brain. Even in adults chelating is indicated for severe chronic lead poisoning. Chelating should not be done in patients with lead colic and is also contraindicated in patients with renal failure. Apart from medical treatment, the source of lead should be identified and removed immediately. Nutritional status of the patient should also be assessed and especially iron deficiency anemia if present should be treated.

5.12 Prevention

Prevention plays the most important role in ensuring children are protected from lead poisoning, Prevention of lead works by eliminating the use of lead and preventing exposure, some of which are listed below.

5.12.1 **Eliminating use:**

a) Phasing out lead in paints

b) Eliminate use of leaded solder in food, drink cans and water pipes.

c) Eliminate the use of lead in schools, school material and toys.

d) Eliminate the use of lead in pottery used in cooking, eating or drinking.

e) Eliminate the use of lead in plumbing fittings

f) Eliminate the use of lead in cosmetics and traditional medications.

g) Wash dust off hands, toys, bottles, windows and floors.

h) Serve food rich in calcium, iron and vitamin C.

5.12.2 **Preventing exposure:**

a) Electric and electronic waste.

b) Safe recycling of lead containing waste.

c) Avoid contaminated sites.

6 Methodology

Type of study:

Community based Case Control study

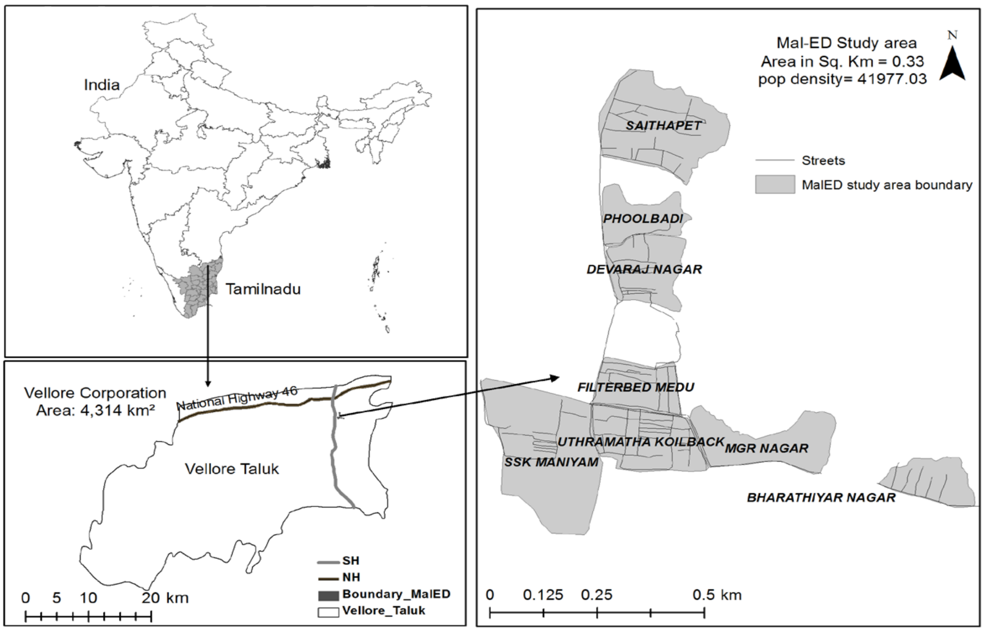
Study setting

The MAL-ED study

The MAL-ED Network established by the Foundation for the National Institutes of Health and the Fogarty International Centre is a multi-country network looking at the complex relationships between childhood malnutrition, enteric infections, gut function and cognitive development. The 8 study sites are epidemiologically and geographically diverse and comprised of low-income populations. The 8 MAL-ED cohort sites are located in Bangladesh, Brazil, India, Nepal, Pakistan, Peru, South Africa, and Tanzania. The study site in India is located in the Vellore, a city in the state of Tamil Nadu. The study site spanned over an area of 0.33 km2 and was distributed over 8 contiguous urban slums with a total population of around 13500. The Christian Medical College (CMC), a not for profit medical institution provides primary and secondary health care services to these urban areas through its Low Cost Effective Care Unit (LCECU), a secondary level hospital located close to the study area.

In the primary study, pregnant women in the area were identified by a door to door survey by a team of field workers and were enrolled in the study after obtaining informed consent. They were followed till childbirth and the babies were enrolled. A total of 251 babies were recruited between March 2010 and February 2012 at an average of 10 babies per month. This birth cohort was followed up till the children attained 36 months of age. Baseline demographic characteristics were collected using a structured questionnaire immediately after childbirth. Monthly anthropometric measurements of the study children, surveillance for illnesses were conducted. Venous blood samples were collected from the enrolled children at 7, 15 and 24 months as per the study protocol and tested for various biomarkers, lead being one of them.The study was conducted in Old town, Salavanpet and neighboring areas in central Vellore where the Mal-ED study is currently been conducted. The participants of the study reside in the urban slums in these areas. The map of the study area is presented in Figure 6.

FIGURE 6: Map of the study area in Vellore city



6.1 Time line:

- January-February 2014: locate the houses of the cases and controls
- March-June 2014: Data collection.
- July-August 2014: Data entry and Analysis

6.2 Participants:

The participants for the study were from the birth cohort of the MAL-ED study whose blood lead levels were estimated at 24 months of age.

Case: Children with BLLs>/=10mcg/dl at 24 months

Control: Children with BLLs<10mcg at 24 months

(Only one child per house was eligible to participate in the study)

6.3 Inclusion and exclusion criteria

Inclusion criteria:

- Children from the Mal-ED cohort who have completed 24 months and whose venous blood has been tested for lead levels at 24 months.
- Children from the Mal-ED cohort who parents were willing to participate in the study.

Exclusion criteria:

- Those participants who had changed residences two months before the 24 month blood sample collection.

6.4 Variables

6.4.1 Outcome variable:

-Blood lead levels (venous blood samples) at 24 months.

6.4.2 Exposure variables:

-Environmental Lead levels

The following variables were measured:

- Socio demographic factors
- Risk factors for elevated BLLs

i) Parental characteristics:

- Education of all the family members.
- Work exposure: high-risk occupations/ contact with chemicals/ duration of work in years/ change of clothes / hygiene.
- Smoking details.

ii) Characteristics of the dwelling place:

- Paint details: house/ roof/ floor/ walls/ windows/ doors
- House renovation
- State of the house
- Cleaning details

iii) Ingestion through drinking water/food/containers/taps/pipes.

iv) Habits and daily activities of the child–Pica/ hand to mouth habits/chewing and licking habits/articles handled by the child.

v) Breast-feeding/ kajal application/ Ayurvedic medication/ toys details.

6.5 Data sources and methods of assessment

6.5.1 **Blood lead levels:**

As per the Mal-Ed study protocol, a maximum of 5 ml of venous blood was collected from the children from veins on the dorsum of the hand at 7, 15 and 24 months for various biochemical tests of which around 1 ml of whole blood was preserved with Dipotassium Ethylenediaminetetraacetic acid (K2 EDTA) for determination of lead levels. The samples were refrigerated immediately and were transported to the lab within a few hours using cold packs. Lead levels in whole blood were estimated by Graphite Furnace Atomic Absorption Spectroscopy (GFAAS) method using an M Series Atomic Absorption Spectrophotometer.(59) The Centers for Disease Control, Atlanta's (CDC) earlier definition of elevated blood lead level (BLL) was 10 μg/dL and above for children under 6 years of age. Recently, the CDC has updated the "level of concern" for BLLs among children to >5 μg/dL (Based on the findings from the National Health and Nutrition Examination Survey (NHANES). Since most of the available information from various similar other studies from India and elsewhere is based on the cut-off value of 10 μg/dL, the same cut-off value has been used in this study to identify elevated BLLs.(4)

6.5.2 **Socio economic status assessed using the "Modified Kuppuswamy's Scale":**

The modified Kuppuswamys scale was used in this study taking into account the education, occupation and income of the head family to classify study groups in to high, middle and low socioeconomic status. The Kuppuswamys scale was developed in 1976 and ever since been updated, as the price index keeps increasing and in this study we used the 2012 modified scale, which was adjusted to 2012s consumer price index.(90)

(A) Education Score		
1	Profession or Honours	7
2	Graduate or post graduate	6
3	Intermediate or post high school diploma	5
4	High school certificate	4
5	Middle school certificate	3
6	Primary school certificate	2
7	Illiterate	1

(B) Occupation Score		
1	Profession	10
2	Semi-Profession	6
3	Clerical, Shop-owner, Farmer	5
4	Skilled worker	4
5	Semi-skilled worker	3
6	Unskilled worker	2
7	Unemployed	1

(C) Monthly family income in Rs		Score	Modified for 1998 [3] in Rs	Modified for 2012 in Rs
1	≥ 2000	12	≥ 13500	≥32050
2	1000-1999	10	6750 - 13499	16020 – 32049
3	750-999	6	5050 - 6749	12020 – 16019
4	500-749	4	3375 - 5049	8010 – 12019
5	300-499	3	2025 - 3374	4810 – 8009
6	101-299	2	676 - 2024	1601 – 4809
7	≤ 100	1	≤ 675	≤ 1600

Total Score	Socioeconomic class
26-29	Upper (I)
16-25	Upper Middle (II)
11-15	Middle/Lower middle (III)
5-10	Lower/Upper lower (IV)
<5	Lower (V)

6.5.3 Determinants for elevated blood lead levels

i) **Parental characteristics:**

- Education of all the family members.
- Work exposure: high-risk occupations/contact with chemicals/duration of work in years/change of clothes /hygiene.
- Smoking details.

ii) **Characteristics of the dwelling place:**

- Paint details: house/roof/floor/walls/windows/doors.
- House renovation
- State of the house
- Cleaning details

iii) **Ingestion** through drinking water/food/containers/taps/pipes.

iv) **Habits and daily activities of the child –**Pica/hand to mouth habits/chewing and licking habits/articles handled by the child.

v) **Breast-feeding/ kajal application/ Ayurvedic medication/ toys details.**

6.6 Sample size:

$$n = \frac{r+1}{r} * \frac{(\bar{p})(1-\bar{p})\left(Z_{\propto/2} + Z_{\beta}\right)^2}{(P_1 - P_2)^2}$$

Where, $\overline{p}$ = mean proportion exposed in cases and controls
$Z_{\propto/2}$ = Significance
Z_{β} = desired power
P_1 = Proportion of cases exposed
P_2 = Proportion of controls exposed
r = ratio of controls to case

- **Prevalence of exposure factor amongst controls = 40%****
- **Expected Odds ratio=2.5**
- **Cases=78**
- **Controls =78**
- **Total=156**

****Vishwanath P, Devegowda D, Prashant A, Nayak N, D'souza V, Venkatesh T, et al. Environmental lead levels in a coastal city of India: The lead burden continues. Indian J Med Sci. 2012 Dec; 66(11-12): 260–6.*

Questionnaire

The questionnaire used was a interviewer administered (Double blinded), Semi structured, pilot tested, Tamil translated and back translated. The questionnaire has been attached in Annexure 1.

The study design was presented to the Christian Medical College- Institutional review Board and the approval to conduct the study was granted after the pilot study.

A pilot study was conducted in a rural village named Thuthipet in Kaniyambadi block to standardize the questionnaire and sample collection technique. After obtaining informed consent, the questionnaire was administered and environmental samples (25 overall) were collected from five houses with pre-school children in the village the consent was taken, the principal investigator administered the questionnaire.

The main study was conducted in Old town, Salavanpet and neighboring slums in central Vellore as shown in Figure 6. Out of the 228 study participants, simple random sampling chose 80 cases and 80 controls and the blinded list was handed over to the principal investigator to visit the houses. Out of the 160 houses only 96 participants were present, while the rest had moved out of the study area or had shifted out of the earlier residence to other houses in the same or neighboring locality at the time of 24 month blood sample collection. In the second step, the blinded list of remaining cases and controls was provided to the principal investigator to visit the houses. A total of 153 participants were available and consented to participate in the study. During the house visits, information sheet was given and consent taken from the parent. The questionnaire was administered to the parent or the primary caregiver of the children along with a detailed study of the daily activity of the child after which drinking water, door paint, wall paint and house paint samples were collected, and based on the activities of the child additional samples were collected if required. The samples were labeled and sent to Vellore Institute of Technology's Total Business Incubator Laboratory for processing and analysis.

FIGURE NO. 7: Detailed diagrammatic Algorithm of the study:

Selection of samples from the Mal-ED study from Old Town/Salavanpet and surrounding areas (one child per house hold)

Visited the houses of cases and controls (Interviewer: blinded)
Based on their blood lead levels –venous blood samples taken at 24 months
(Controls=BLLs<10mcg and cases =BLLs>/=10mcg/dl)

Information sheet given and Consent taken

Administration of the Questionnaire to the parents of cases and controls

Detailed daily activities of each child studied

Environmental samples collected from the surroundings of each participant:

5 environmental samples or more will be collected for the child's immediate surrounding

- 4 Compulsory samples: dust, water, paint from walls windows and doors
- 1 or more samples based on child's daily activities (eg: surma/paint from grill gate/ toys)

Samples processed and analyzed in Vellore Institute of Technology

Data entry analysis and submission of results

6.6.1 Environmental sources of lead:

6.6.1.1 Environmental Lead estimation methods

In this study we used the Flame atomic absorption spectrometry (FAAS) method to estimate the environmental lead levels and the instrument used to estimate lead in samples is the AAS Varian Spectra A 240 absorption lamps with respect to metal ions in the aqueous solution.

6.6.2 Sample collection and estimation of lead levels

6.6.2.1.1 **Paint**: The samples were scrapped from various surfaces like walls, windows, doors, grills and beds using a blade/ paint scraper on to a cardboard piece which was discarded after each sample collection to avoid cross contamination .The samples were then put into cleaned labeled glass bottles. After which the sample details were entered in the questionnaire with the unique sample ID number.

6.6.2.1.2 **House dust:** The house dust was collected on a new cardboard piece after sweeping the rooms of the house with the household broom and the sample was emptied intocleaned labeled glass bottles.

6.6.2.1.3 **Toys:** If possible the entire toy was collected. But if the participant was hesitant to give the toy, a piece of the toy especially the area bitten by the child was cut and labeled.

6.6.2.1.4 **Drinking water:** Drinking water was collected in Cleaned 50 ml containers.

The solid samples were collected in 5 ml glass bottles with rubber caps which were washed and cleaned prior to collection. The drinking water samples were collected in 50 ml plastic bottles which were cleaned prior to sampling to avoid cross contamination. The samples were transported at the earliest under field conditions to the testing laboratory, a nationally standardized Biochemistry lab in Vellore Institute of Technology-TBI Laboratory (a joint initiative by VIT University and Department of Science and Technology (DST), Govt. of India) for further processing and testing. In this study lead levels in the environmental samples were estimated using Flame atomic absorption spectrometry (FAAS) technique at the Biochemistry lab in VIT, Vellore.

The solid samples (paint, house dust, cosmetics, and smaller toys) were first weighed in a calibrated standardized weighing scale. The paint from the larger toys was scraped and the paint was weighed. The weighed samples were then mixed with nitric acid and heated on a heating mantel till the sample dissolved in the acid. The amount of nitric acid added to dissolve the sample was also noted. The sample was heated till the entire sample dissolve the acid and the time taken was noted. The dissolved sample was then filtered and transferred into a 100 ml conical flask and diluted to 100 ml with distilled water. Ten ml of the diluted solution was then taken in a labeled test tube and was sent for analysis. Similarly 10 ml of the drinking water samples were directly sent to the lab for analysis. The lead levels in the samples were reported in Parts per million (PPM).

6.7 Analysis

6.7.1 Data entry:

The data was entered using Epi Info version 7 developed by CDC USA.

6.7.2 Data analysis:

The data was analyzed using SPSS (Statistics Package for Social Sciences) software package version 16 licensed to Christian Medical College, Vellore.

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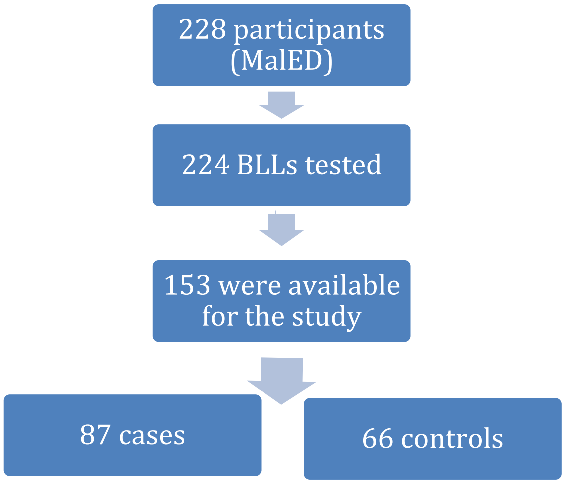
7 Results

7.1 Blood lead levels among the study participants

From the cohort of 224 children, cases and controls were selected by simple random sampling. Children with BLL more or equal to 10 mcg/dl were considered as ‘cases’ and children with BLLs up to 9 mcg/dl were considered as ‘controls’ for this study. A total of 87 cases (BLL>=10mcg/dl) and 66 controls (<9mcg/dl) were included for analysis.

FIGURE 8: Flowchart illustrating the numbers enrolled



The following table describes the characteristic in BLLs among cases and controls

TABLE 2: Mean, Median, Standard deviation and range of BLLs among cases and controls

	Cases	Controls
Mean (SD)	16.6(9.4)	7.4(1.9)
Median	13.7	7.9
Inter quartile range	11.2-17.1	6.2-8.9
Minimum	10	1.5
Maximum	66.8	9.9

The mean(SD) BLLs were 16.6 (9.4) and 7.4 (1.9) among the cases and controls respectively, ranging from 10 to 66.8 in cases and 1.5 to 9.9 among controls.

7.2 Socio-demographic characteristics of the study participants

The following table describes the distribution of various socio-demographic characteristics of the study participants like gender, age, religion, socio-economic scale, family type, total people or children per house and number of rooms among cases and controls.

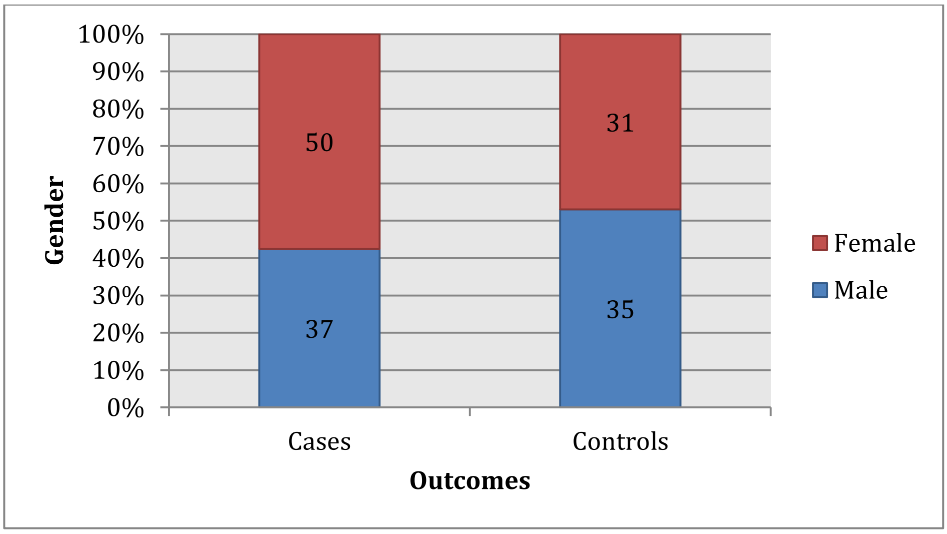
TABLE 3: Base line socio-demographic characteristics

N=153	Variable	Cases n (%) N=87	Control n (%) N=66	Total n (%)
GENDER	Male	37 (42.5)	35(53)	72(47.1)
	Female	50(57.5)	31(47)	81(52.9)
AGE IN MONTHS	25-29	12 (13.8)	5 (7.6)	17 (11.1)
	30-34	27 (31)	8 (12.1)	35 (22.9)
	35-39	16(18.4)	12 (18.2)	28 (18.3)
	40-44	18 (20.7)	23 (34.8)	41(26.8)
	45-49	14 (16.1)	14 (21.2)	28 (27.3)
	50-55	0 (0)	4 (6.1)	4 (2.6)
RELIGION	Christian	10 (11.5)	6 (9.1)	16(10.5)
	Hindu	55(63.2)	48 (72.7)	103 (67.3)
	Muslim	22 (25.3)	12 (18.2)	34 (22.2)
SES CLASSIFICATION	Upper middle	7(8)	7(10.6)	14(9.2)
	Lower middle	31(35.6)	19(28.8)	50(32.7)
	Upper lower	49(56.3)	40(60.6)	89(58.2)
FAMILY TYPE	Extended	38 (43.7)	33 (50)	71 (46.4)
	Joint	14 (16.1)	7 (10.6)	21 (13.7)
	Nuclear	35 (40.2)	26 (39.4)	61 (39.9)
TOTAL PEOPLE PER HOUSE	<=4	20(23)	12 (18.2)	32 (20.9)
	5 TO 8	50 (57.5)	48 (72.7)	98 (64.1)
	>=9	17 (19.5)	6 (9.1)	23 (15)
TOTAL CHILDREN PER HOUSE	1	10 (11.5)	10 (15.4)	20 (13.2)
	2	37 (42.5)	20 (30.8)	57 (37.5)
	3	20 (23)	24 (36.9)	44 (28.9)
	4	10 (11.5)	8 (12.3)	18 (11.8)
	>=5	10 (11.5)	3 (4.6)	13 (8.6)
NUMBER OF ROOMS PER HOUSE	1	17 (19.5)	13 (19.7)	30 (19.6)
	2	35 (40.2)	25 (37.9)	60 (39.2)
	3	18 (20.7)	16 (24.2)	34 (22.2)
	4	11 (12.6)	9 (13.6)	20(13.1)
	>=5	6 (6.9)	3 (4.5)	9 (5.9)

7.2.1 Gender distribution among the study participants

The distribution of males and females among cases was 43% (37/87) and 57.5% (50/87) respectively; this distribution among controls was 53% (35/66) and 47% (47/66) respectively. The association between gender and elevated BLLs were not statistically significant. (Chi square value1.66, p-value>0.05; OR=0.66, 95%CI=0.34-1.25).

FIGURE 9: Gender distribution among cases and controls



7.2.2. Child characteristics

7.2.2.1. Breast feeding practices

Among the 153 participants the mean (SD) and median (IQR) duration of exclusive breast feeding was 4.8(1.9) and 5 (1-7.5) months respectively, ranging from 0 to 9 months. The WHO recommended mean of 6 months was chosen as the cutoff for the risk factor analysis.

TABLE NO 4. Exclusive breast-feeding and association with BLLs

EBF	Cases n (%)	Controls n (%)	Total n (%)
<6 Months	49 (56.3)	30 (45.5)	79 (51.6)
>=6 Months	38 (43.7)	36 (54.5)	74 (48.4)
Total	87	66	153

Among the 153 participants, 51.6% were exclusively breast fed for less than 6 months. Fifty six percent (49/87) of the cases were exclusively breast fed for less than 6 months as compared to 45.5% (30/66) of the controls. Seventy-four (48.4%) participants were exclusively breast fed for 6 months or more. Even though the percentage of participants among cases who were exclusively breast fed for less than 6 months was higher, there was no statistically significant association between duration of exclusive breast feeding and elevated blood level levels. (Chi sq. value=1.78, p value>0.05; OR=1.55, 95% CI=0.81-2.95).

7.2.2.2.Birth weight distribution among the study participants

TABLE 5: Low birth weight and association with elevated BLLs

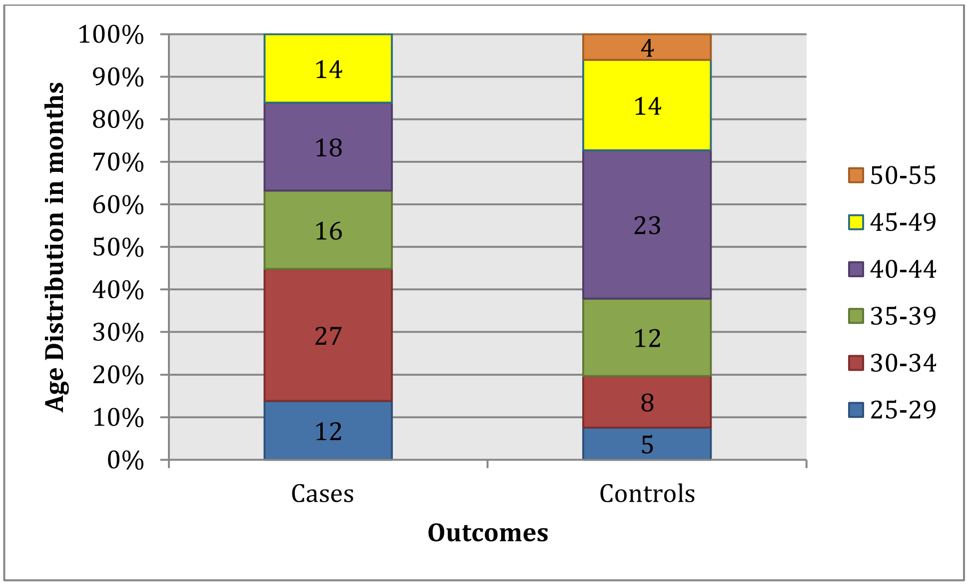
Birth weight	Cases n (%)	Controls n(%)	Total n (%)
Low birth weight	9 (10.8)	15 (23.1)	24 (16.2)
Normal	74 (89.2)	50 (76.9)	124 (83.8)
Total	83	65	148

The WHO recommended low birth weight of <2.5 kg was chosen as the cutoff for the risk factor analysis. Eleven percent (9/83) of the cases were born low birth weight as compared to 23.1% (15/65) of the controls.(Chi sq. value=4.02, p-value =0.045; OR=0.405, 95% CI=0.17-0.99).

7.2.3. Age distribution of the study participants

During the time of interview, the mean (SD) and median age (IQR) of the study participants were 39.3 (7.0) and 39.6 (32.75-45.52) months respectively, ages ranged from 26 to 52 months. The age group wise distribution of cases and controls are shown in Figure 10.

FIGURE 10: Age distribution at interview in months (both cases and controls)



7.2.4. Religion

Majority of the cases (63%) and controls (72.7%) belonged to the Hindu religion followed by Muslims and Christians.

The religion wise breakup of the study population is presented in figure 11.

FIGURE 11: Religion of the cases and controls

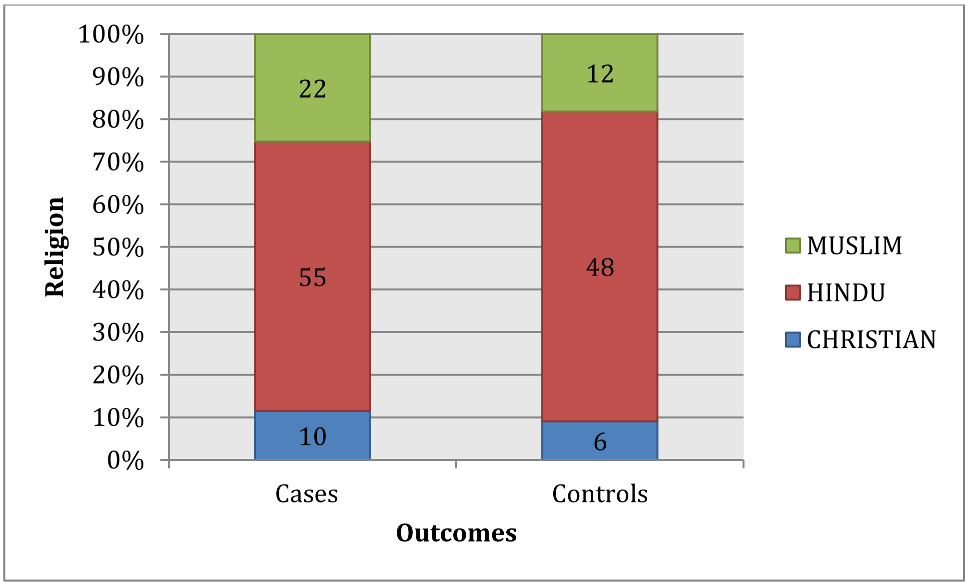


TABLE 6: Religion and association with BLLs

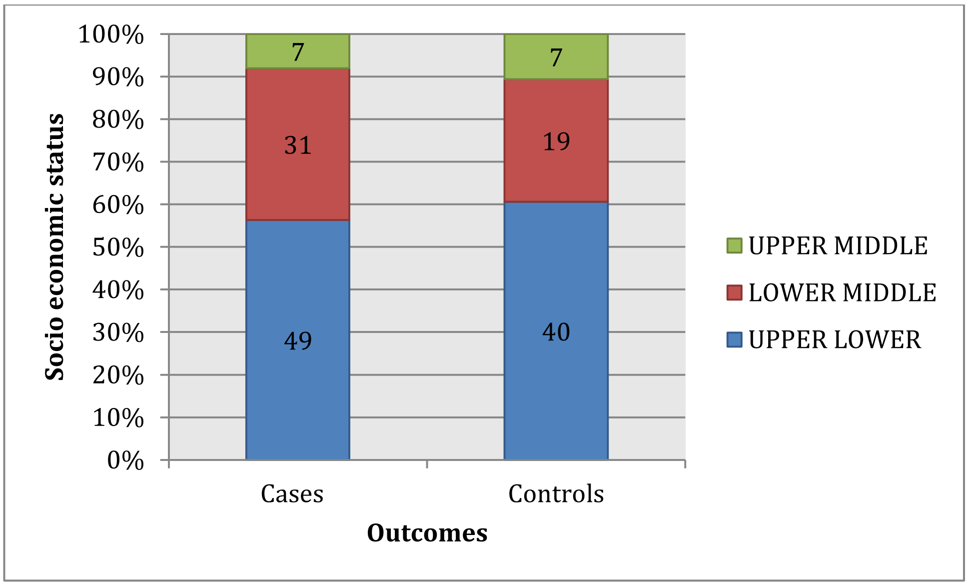
Religion	Cases n (%)	Controls n (%)	Total n (%)
Muslim	22 (25.3)	12 (18.2)	34 (22.2)
Others	65 (74.7)	54 (81.8)	119 (77.8)
	87	66	153

Among the 153 subjects, 22% of the subjects belonged to the Muslim religion. Twenty five percent (22/87) of the subjects were cases as compared to 18% (12/66) of the controls. Though the percentages of Muslims were higher among cases there was no statistically significant association.(Chi sq. value=1.10, p-value =0.295; OR=1.52, 95% CI=0.69-3.36).

7.2.5. Socio–economic status of the study participants

The Socio economic classification of the study participants based on the modified Kuppuswamys SES scale is as follows:

FIGURE 12: Socio-economic classification of the participants

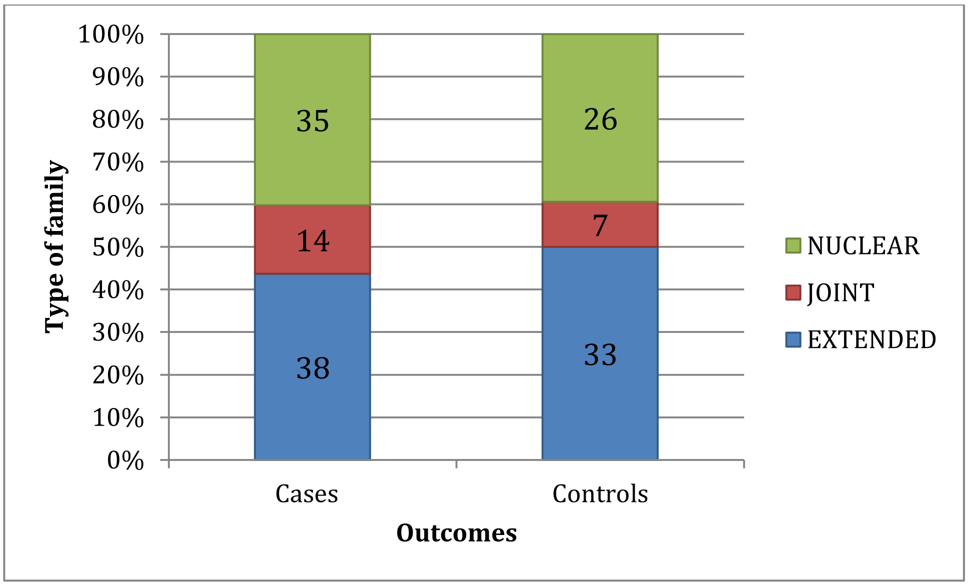


Among the 153 participants majority of them, 89(58.2%) belonged to the upper-lower class. Among the cases, 56.3%, 35.6% and 8% belonged to the upper lower, lower middle and upper middle socio-economic strata respectively while the proportions were 60.6%, 28.8% & 10.6% among the controls. Socio-economic status of the family was not significantly associated with elevated BLLs among the study participants (Chi square for trend: 0.925, p value>0.05).

7.2.6. Type of family

The dominant type of family among cases was extended families (43.7%) followed by nuclear (40.3) and joint families (16.1%). This pattern was consistent among controls as well.

FIGURE 13: Type of family among cases and controls



7.2.7.Number of members in the household and association with EBLLs

In this study, the mean (SD) and median (IQR) number of members in the household was 6.3 (2.1) and 6 (5-7) people respectively and the family size ranged from 2 to 13 people. The control median of 6 people per family was used to dichotomize for risk factor analysis.

TABLE 7: Number of persons living in each family

No. of people	Cases n (%)	Controls n (%)	Total n (%)
>=6	52 (59.8)	35 (53)	87 (56.9)
<6	35 (40.2)	31 (47)	66 (43.1)
Total	87	66	153

Among the cases, 59.8% (52/87) had more than or equal to 6 people living in the house and 40.2% (35/87) had 2 to 5 people living per house. Similarly among the controls, 53% (35/66) had 6 to 13 people living in the house and 47% (31/66) had 2 to 5 people living per house. Although the percentage of cases living in homes with 6 to 10 people was higher, the was no statistical association with elevated BLLs. (Chi square value= 0.70, p value >0.05; OR= 1.32, 95%CI= 0.69-2.51).

7.2.8. Number of children living in each household and association with EBLLs

In this study the mean (SD) and median (IQR) children in each family are 2.7(1.26) and 2 (2-3) children respectively, ranging from 1 to 8 children. The control median of 3 children was chosen as the cutoff for Univariate analysis.

TABLE 8: Number of children living in each family

No. of children	Cases n (%)	Controls n (%)	Total n (%)
>=3	40 (46)	35 (53.8)	75 (49.3)
< 3	47 (54)	30 (46.2)	77 (50.7)
	87	65	152

Among the cases, there were 46 % (40/87) with 3 or more children in the family and 54% (47/87) participants had less than 3 children in the family. Among the controls, there were 53.8% (35/65) with 3 or more children in the family and 46.2% (30/65) participants had 2 or less children in the family. There was no statistically significant association between number of children in the family and elevated BLLs (Chi square value=0.92, p-value >0.05; OR= 0.73, 95%CI= 0.38-1.39).

7.2.9. Child death in the family and association with EBLLs

TABLE 9: Child death in the family and association with EBLLs

Child death in the family	Cases n (%)	Controls n (%)	Total n (%)
Yes	10 (11.5)	4 (6.1)	14 (9.2)
No	77 (88.5)	62 (93.9)	139 (90.8)
Total	87	66	153

Eleven percent (10/87) of the case families had a child death in the past as compared to 6.1% (4/66) of the controls. Although the percentage family's with child death was higher among cases (11.5% vs 6.1%), this finding was not statistically significant. (Fisher's exact test p-value >0.05; OR=2.01, 95%CI=0.60-6.73).

7.3. Occupational exposure among family members

Father's occupation:

TABLE 10: List of occupations of the father among cases and controls

High risk occupations	Cases n (%)	Controls n (%)	Total n (%)
Ac mechanic	0 (0)	1(7.7)	1 (3.1)
Automobile spare shop	0 (0)	1 (7.7)	1 (3.1)
Building demolisher	2(10.5)	0 (0)	2(6.3)
Cell service	1 (5.3)	0(0)	1(3.1)
Contractor	0 (0)	1 (7.7)	1 (3.1)
Cycle shop	0 (0)	1 (7.7)	1 (3.1)
Door polisher	0 (0)	1 (7.7)	1 (3.1)
Mason	5 (26.3)	3 (23.1)	8 (25)
Mechanic	1 (5.3)	1 (7.7)	2 (6.3)
Mobile service	1 (5.3)	0 (0)	1 (3.1)
Painter	6 (31.6)	3 (23.1)	9 (28.1)
Plumber	1 (5.3)	0 (0)	1 (3.1)
Printer	1 (5.3)	0 (0)	1 (3.1)
Tyre shop	1 (5.3)	0 (0)	1 (3.1)
Welder	0 (0)	1 (7.7)	1 (3.1)
Total	19	13	32 (100)
Non High Risk Occupations			
Beedi roller	5 (7.9)	10 (21.3)	15 (13.6)
Shop keepers	3 (4.8)	1 (2.1)	4 (3.6)
Hotel industry	24 (38.1)	18 (38.3)	42 (38.2)
Driver	1 (1.6)	3 (6.4)	4 (3.6)
Daily wage worker	16 (25.4)	9 (19.1)	25 (22.7)
Others	14 (22.2)	6 (12.8)	20 (18.2)
Total	63	47	110 (100)

Among the 32 fathers of the participants involved with high risk occupation, majority 9 (28.1%) of them were Painters followed by 8 (25%) construction workers followed by Automobile mechanics 2 (6.3%) and building demolishers 2 (6.3%). 11 fathers did not stay with the participants. The detailed breakdown of paternal occupation is presented in Table 10.

Among the 142 working fathers of the participants 77.5% (110/142) worked in Non high risk occupations. Most of them (38.2%) worked in the

Hotel industry followed by (22.7%) daily wage work who mainly involved working in the market as load lifters.

TABLE 11: High risk occupation of the father and BLLs

High risk occupation	Cases n (%)	Control n (%)	Total n (%)
Yes	19(23.2)	13(21.7)	32 (22.5)
No	63(76.8)	47(78.3)	110 (77.5)
Total	82	60	142

Among the cases, 23% (19/82) fathers were involved in high-risk occupations and among the controls 22 %(13/60) worked in High risk occupations. The remaining 110 (77.5%) worked in non high-risk occupations. Even though the percentage of father's occupations working in high-risk areas was higher among cases, there was no statistically significant association between fathers' high risk occupation and elevated BLLs among the study participants at 24 months of age (Chi sq. value=0.045, p-value >0.05; OR=1.09, 95%CI=0.49-2.43).

Number of earning members in the family with high-risk occupations

TABLE 12: Number of earning members in the family with high-risk occupations

High risk earning members	Cases n(%)	Controls n (%)	Total n(%)
3	2 (2.3)	0 (0)	2 (1.3)
2	2 (2.3)	4 (6.1)	6 (3.9)
1	17 (19.5)	14 (21.2)	30 (20.3)
0	67 (75.9)	48 (72.7)	115 (74.5)
	87	66	153

Among the 153 participants, 25.5% of the households had more than or equal to 1 earning member in the family working in a high-risk occupation. The distribution was almost same among cases and controls.(Chi square for trend: 1.2 p –value >0.05)

Mother's occupation:

TABLE 13: List of occupations of the mother

Mothers' Occupation	Cases n (%)	Controls n (%)	Total n (%)
HOUSE WIFE	69 (81.2)	48 (77.4)	117 (79.6)
MAID	4(4.7)	4 (6.5)	8 (5.4)
BEEDI WORKERS	6 (7.1)	3 (4.8)	9 (6.1)
OTHERS	6 (7.1)	7 (11.3)	13 (8.8)
Total	85	62	147

Among mothers of the participants, none of them were involved in high-risk occupations, majority of them 117 (79.6%) were housewife's, 9 (6.1%) were beedi workers, 8 (5.4%) were working as housemaids and the rest of the 13 (8.8%) were involved in other occupations.

7.4 Housing characteristics and association with EBLLs

7.4.1 Number of rooms and association with EBLLs

In this study the mean (SD) and median (IQR) rooms in each house are 2.6 (1.4) and 2 (2-3) respectively, ranging from 1 to 7 rooms. The control median of 2 rooms was used as a cutoff for risk factor analysis.

TABLE 14: Number of rooms per house and association with EBLLs

No. of rooms	Cases n (%)	Controls n (%)	Total n (%)
1 to 2	52 (59.8)	38 (57.6)	90 (58.8)
3 to 7	35 (40.2)	28 (42.4)	63 (41.2)
Total	87	66	153

Among the 153 participants, 60% (52/87) cases lived in 1 to 2 rooms and 58% (38/66) of the controls lived in 1 to 2 rooms. The remaining 63 (41.2%) family's lived in 3 to 7 rooms per house. Although the percentage of cases living in homes with 1 to 2 rooms was higher, there was no statistical association with elevated BLLs. (Chi sq. value=0.075, p-value>0.05; OR=1.09, 95%CI=0.57-2.10).

7.4.2. Number of people per room and association with EBLLs

In this study the mean (SD) and median (IQR) people per room in each house are 3 (1.5) and 2.5 (1.88-4) respectively, ranging from 1 to 9 people per room. The control median of 3 people per room was chosen as the cut off for risk factor analysis.

TABLE 15: Number of people per room associated with BLLs

People /room	Cases n (%)	Controls n (%)	Total n (%)
>=3	52(59.8)	37 (56.1)	89 (58.2)
<3	35 (40.2)	29 (43.9)	64 (41.8)
Total	87	66	153

Among the 153 participants, 59.8% (52/87) family's of cases had more or equal to 3 people per room in the house, and 56.1%(37/66) were controls.

The remaining lived in families with <2 people per room in each house. Although the percentage of cases living in homes with > =3 people per room was higher, the was no statistical association with elevated BLLs. (Chi sq value=0.21, p-value >0.05; OR=1.16, 95%CI=0.61-2.23).

7.4.3 Type of roof and association with EBLLs

Majority of the study houses had concrete roofs (49.7%), followed by asbestos sheets (22.9%) and terracotta tiles (17%). Asbestos and tin roofs were established risk factors chosen for the univariate analysis of this risk factor. The distributions of types of roof are presented in Figure 14.

FIGURE 14: Type of roof among cases and controls

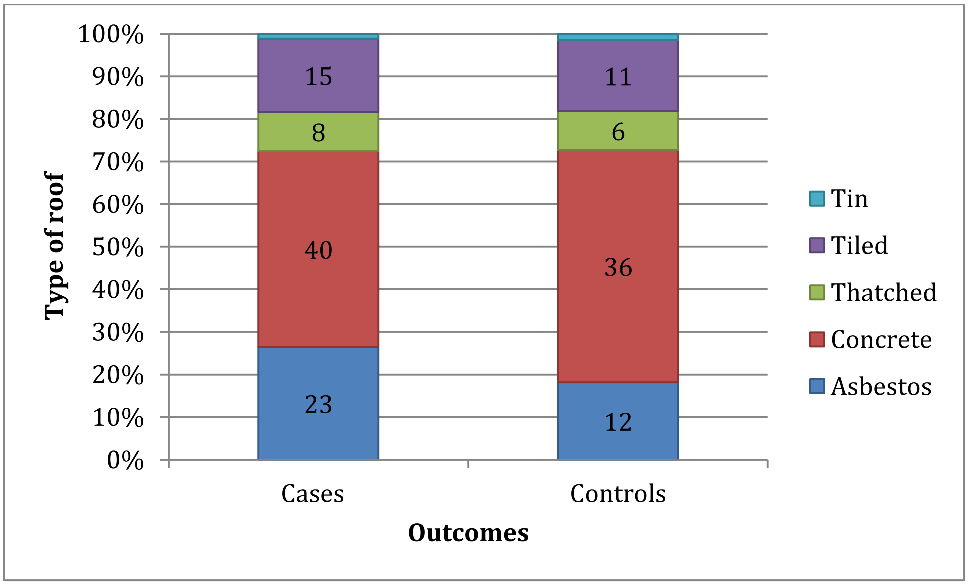


TABLE 16: Type of roof associated with elevated BLLs

Type of roof	Cases n (%)	Controls n (%)	Total n (%)
Asbestos/tin	23 (26.4)	12 (18.2)	35 (22.9)
Others	64 (73.6)	54 (81.8)	118 (77.1)
Total	87	66	153

Among the cases 26.4% (23/87) lived in houses with roofs made of asbestos or tin and 73.6% (64/87) lived under other roofs. And among the controls 18.2 % (12/66) lived under roofs made of asbestos or tin and 81.8% (54/66) lived under other roofs. There was no statistically significant association between type of roof and elevated BLLs. (Chi sq. value=1.45, p-value >0.05; OR=1.62, 95% CI=0.74-3.55).

7.4.4.Type of floor and association with EBLLs

TABLE 17: Type of floor associated with BLLs

Type of floor	Cases n (%)	Controls n (%)	Total n (%)
Earth/mud/clay	7(8)	2 (3)	9 (5.9)
Others	80 (93)	64 (97)	144 (94.7)
Total	87	66	153

Among the 153 participants, 144 (94.7%) stayed in houses with concrete or tiled floors and the remaining stayed in houses with earth mud or clay floors. Among the Cases 8% (7/87) and controls 3% (2/66) lived in houses with mud floors. Although the percentage of cases living in houses with earth/mud/clay was higher, there was no statistically significant association with elevated BLLs. (Chi sq. value=1.705, p-value>0.05; OR=2.8, 95%CI=0.56-13.95).

7.4.5.Type of walls of the house and association with EBLLs

TABLE 18: Type of wall associated with BLLs

Type of wall	**Cases n (%)**	**Controls n (%)**	**Total n (%)**
Mud	19 (21.8)	8 (12.1)	27 (17.6)
Others	68 (78.2)	58 (87.9)	126 (82.4)
Total	87	66	153

Among the cases 22% (19/87) and controls 12% (8/66) lived in houses with mud floors. Although the percentage of cases living in houses with earth/mud/clay was higher, there was no statistically significant association with elevated BLLs. (Chi sq. value=2.44, p-value >0.05; OR=2.03, 95% CI=0.83-4.97).

7.4.6.Type of paint used on the walls and association with EBLLs

FIGURE 15: Types of wall paints used in participant households

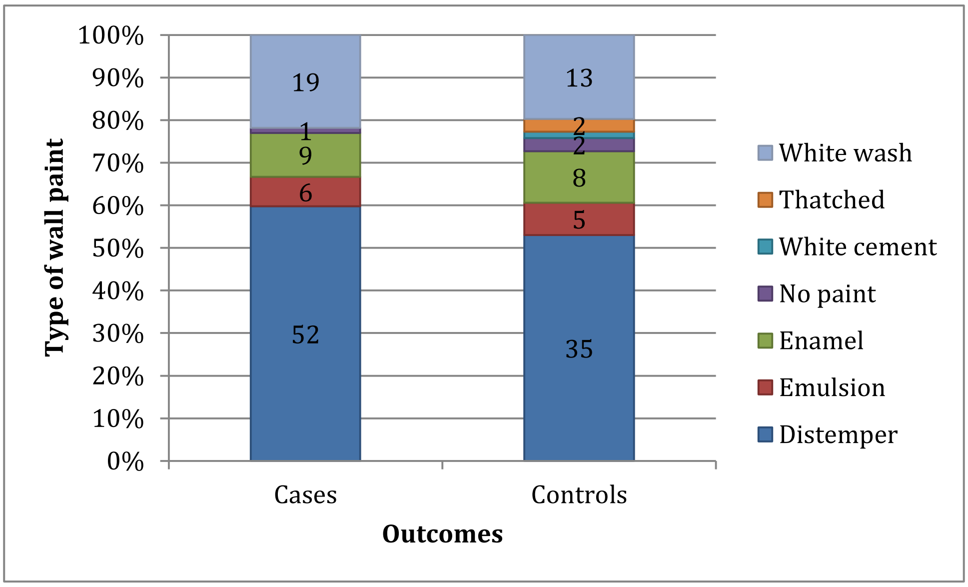


TABLE 19: Type of wall paint associated with BLLs

Type of wall paint	Cases n (%)	Controls n (%)	Total n (%)
Emulsion/enamel	15 (17.4)	13 (21.3)	28 (19)
Whitewash/distemper	71 (82.6)	48 (78.7)	119 (81)
Total	86	61	147

Most of the houses (81%) were painted with distemper or white washed and the remaining were painted with emulsion or enamel paints. The distribution of various paints among cases and controls are described in Figure 15. Emulsion and enamel paints were established risk factors for elevated BLLs and chosen for Univariate analysis. Among the cases, 17.4% (15/87) lived in houses painted with enamel and emulsion while 21% (13/61) of the controls also stayed in similar houses. There was no statistically significant association between type of paint and elevated BLLs. (Chi sq. value=0.35, p-value >0.05; OR=1.28, 95% CI=0.56-2.93).

7.4.7. Number of times the house was painted in the last 5 years

Among the 147 participant houses were painted the last 5 years the mean and median (IQR) were 1.9(1.6) and 1 (1-3) times respectively, ranging from 0 to 9 times. Once or more times painted in the last 5 years were chosen as a risk factor to dichotomize the data for Univariate analysis.

TABLE 20: Number of times house was painted in the last 5 years and association with EBLLs

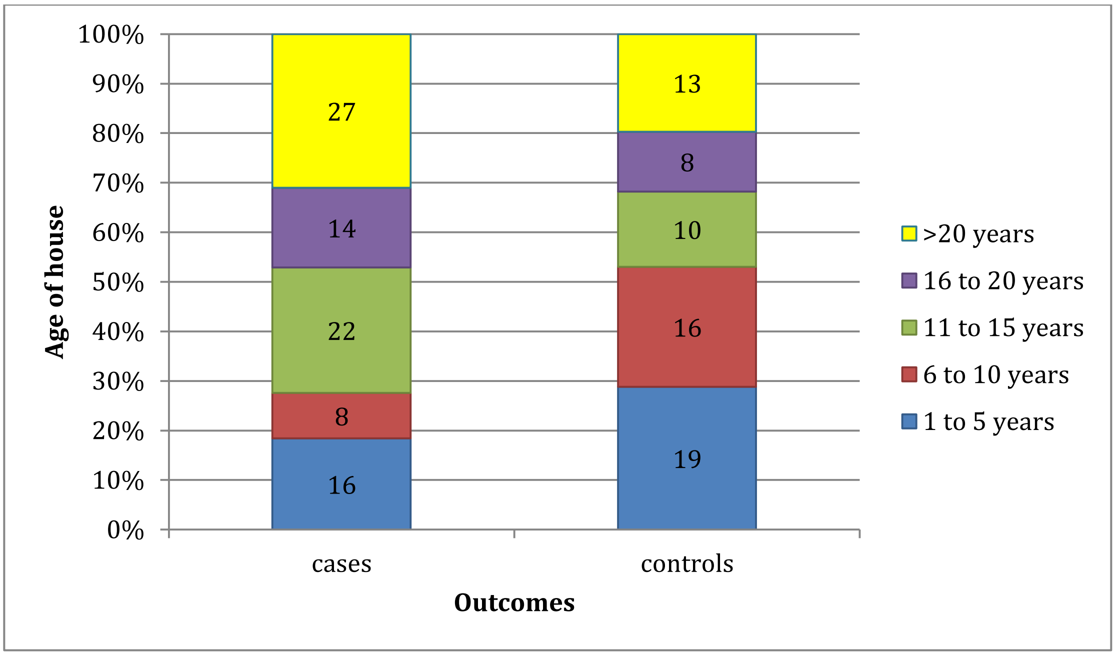
Times house painted last 5 years	Cases n (%)	Controls n (%)	Total n (%)
>=1	80 (93)	49 (80.3)	129 (87.8)
Nil	6 (7)	12 (19.7)	18 (12.2)
Total	86	61	147

Majority of the houses 129 (87.8%) had been painted more than once in the last 5 years. Ninety three percent (80/86) of the case houses and 80% (49/61) of the controls houses were painted more than once in the last 5 years. The remaining 18 (12.2%) houses were not painted. The house being painted twice or more times over the last 5 years was significantly associated with elevated BLLs. (Chi sq. value=5.35, p-value= 0.02; OR=3.27, 95% CI=1.5-9.26).

7.4.8.Age of the house and association with EBLLs

Among the 153 participants the mean (SD) and median (IQR) age of the houses were 15 (11.6) and 14 (6-20) years respectively, ranging from 1 to 60 years. The control median of >= 10 years was chosen as the cutoff to dichotomize the data for Univariate analysis. The distribution of the age of houses among cases and controls are explained in Figure 16.

FIGURE 16: Age of the houses among the participants



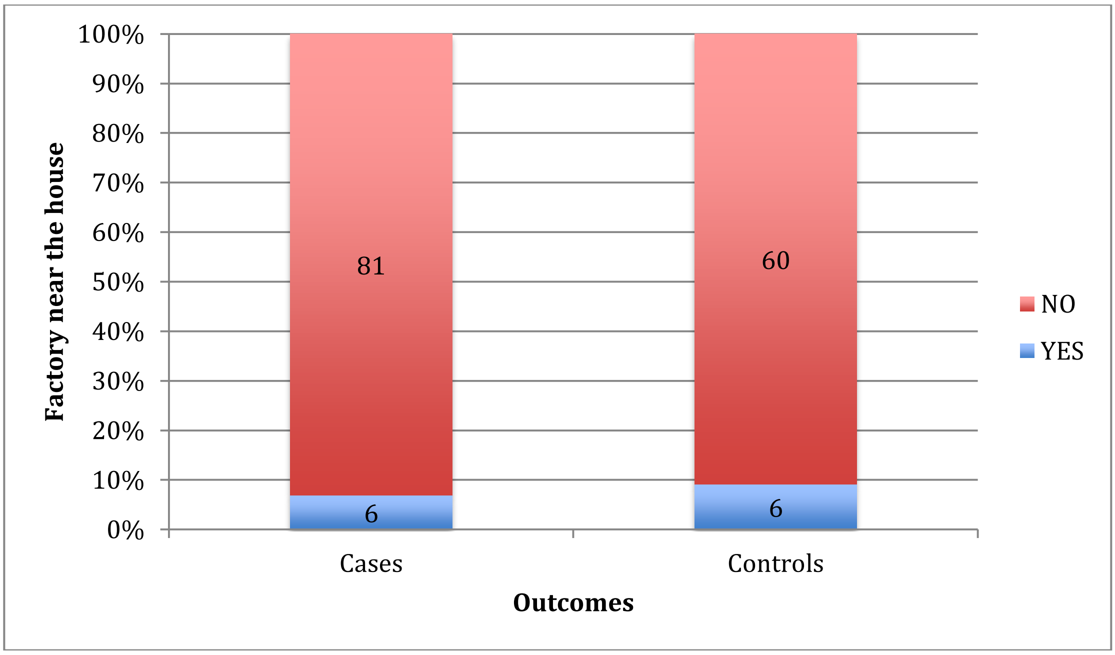
Eighty one percent (70/87) of the cases and 56% (37/66) of controls lived in houses built more than or equal to 10 years. The remaining 30% (46 /153) lived in houses aged less than 10 years old. There was a significant association between age of the house >=10 years and elevated BLLs. (Chi sq. value=10.63, p-value= 0.001; OR=3.23, 95% CI=1.57-6.63) as shown in Table 10.4.9.1

TABLE 21: Age of the house associated with BLLs

Age of the house in years	Cases n (%)	Controls n (%)	Total n (%)
>=10	70 (80.5)	37 (56.1)	107 (69.9)
<10	17 (19.5)	29 (43.9)	46 (30.1)
Total	87	66	153

7.4.9. Location of any Industry or factory near the house and association with EBLLs

FIGURE 17: Factory near the house among cases and controls

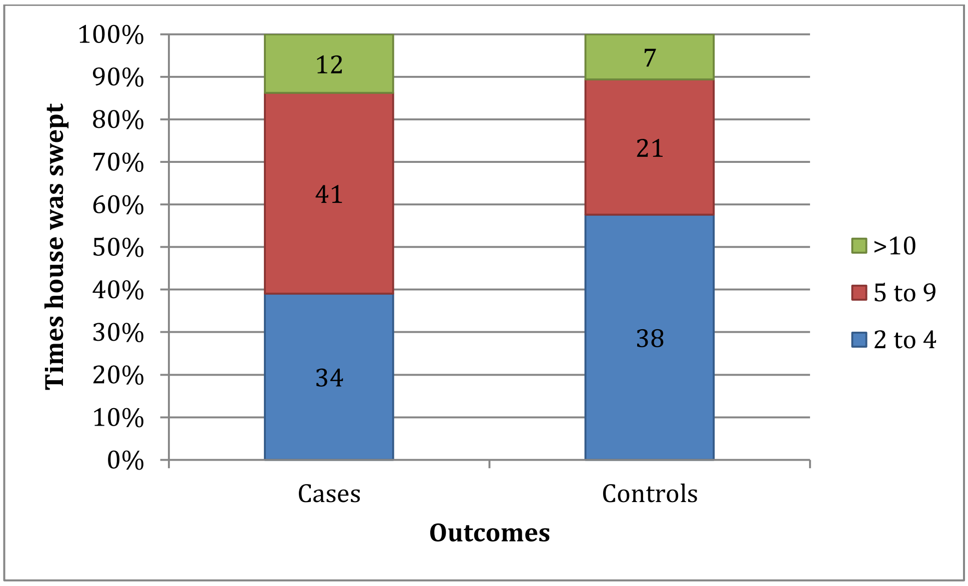


Among the 153 participants 12 participants had factories, small-scale industries near their house. Six point nine percent (6/87) of the cases and 9.1%(6/66) of the controls had factories, small-scale industries near their house. The remaining 141 (92.2%) did not have any factories near their houses. There was no statistically significant association between this risk factor and elevated BLLs. (Chi sq. value=0.25, p-value>0.05; OR=0.74, 95% CI=0.22-2.41).

7.4.10.House sweeping practices and association with EBLLs

Among the 153 participants, the mean (SD) and median (IQR) times the house was swept 5 (2.5) and 4.5 (3-6) times per day respectively, ranging from 2 to 15 times. The control median of 4 times per day was chosen as the cutoff for risk factor analysis.

FIGURE 18: Number of times the house was swept associated with BLLs



Fifty one percent (44/87) of the cases and 68.2% (45/66) of the controls swept the house less than 4 times per day. The remaining 41.8% participant houses swept the house more than or equal to 5 times per day. There was no significant association between the house being swept more than4 times and elevated BLLs. (Chi sq. value=2.89, p-value= 0.09; OR=1.81, 95% CI=0.91-3.52).

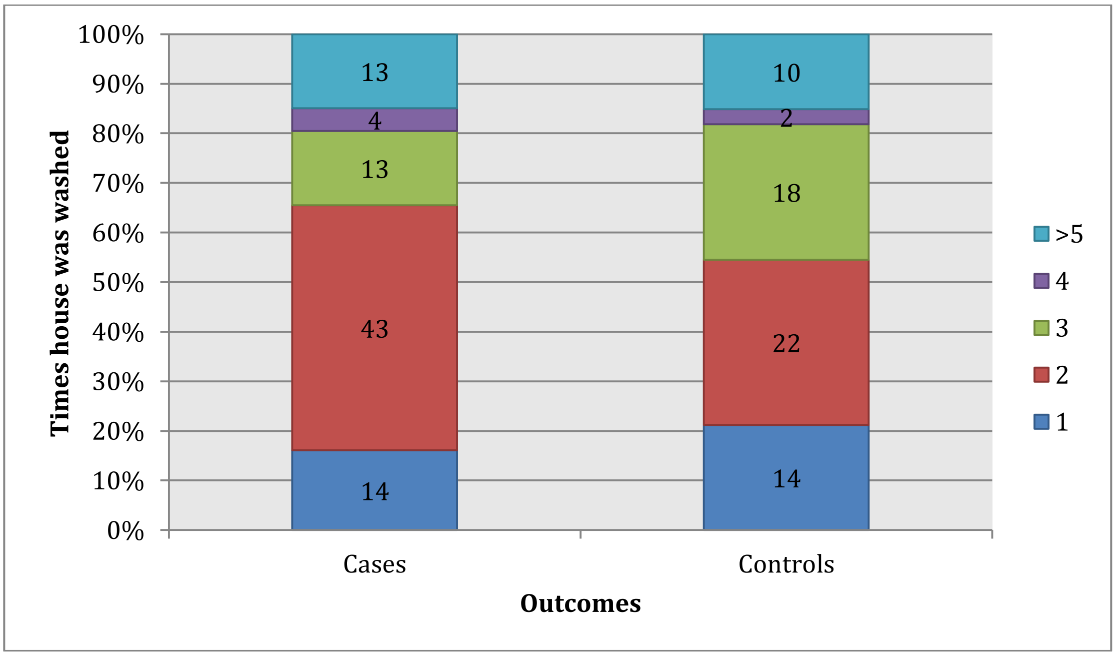
TABLE 22: Times swept per day and association with BLLs

Times swept per day	Cases n (%)	Controls n (%)	Total n (%)
>=4	64 (73.6)	40 (60.6)	104 (68)
<4	23 (26.4)	26 (39.4)	49 (32)
Total	87	66	153

7.4.11.House washing practices and association with EBLLs

Among the 153 participants the mean (SD) and median (IQR) number of times the house washed in a week was 3.2 (4.3) and 2 (2-3) respectively, ranging from 1 to 49 times per week**.** The control median of 2 times per week was chosen as the cutoff for risk factor analysis. The distribution of house washing practice are described in Figure 19.

FIGURE 19 Number of times the house was washed per week and EBLLs



Majority of the households, 60.8% swept their houses 1 to 2 times per week. Sixty five percent (57/87) of the cases swept the houses 1-2 times per week as compared to 55% (36/66) of the controls. The remaining 39.2% households swept more than 3 times per week. Although the percentage of houses swept less than 1 to 2 times was higher among cases, there was no statistically significant association with elevated BLLs. (Chi sq. value=1.90, p-value >0.05; OR=1.58, 95% CI=0.82-3.05).

7.5 Habits of the children and association with EBLLs

TABLE 23: Habits of the study participants in both cases and control

Habit		Cases -n (%)	Controls -n (%)	Total- n (%)	Chi- square p value	Odds Ratio	95 % CI	
							Lower	Upper
LICK/CHEWS PAINT	Present	24 (27.6)	11 (16.7)	35 (22.9)	0.11	1.91	0.86	4.24
	Absent	63 (72.4)	55 (83.3)	118 (77.1)				
EATS MUD	Present	15(17.2)	9 (13.6)	24 (15.7)	0.54	1.32	0.54	3.23
	Absent	72 (82.8)	57 (86.4)	129 (84.3)				
CHEWS DOORS/GRILLS/SILLS	Present	33 (37.9)	26 (39.4)	59 (38.6)	0.85	0.94	0.49	1.81
	Absent	54(62.1)	40 (60.6)	94 (61.4)				
CHEWS PENCILS/CRAYONS	Present	29 (33.3)	22 (33.3)	51 (33.3)	1	1	0.51	1.97
	Absent	58 (66.7)	44 (66.7)	102 (66.7)				
CHEWS CHALK	Present	4 (4.6)	7 (10.6)	11 (7.2)	0.15	0.41	0.11	1.45
	Absent	83 (95.4)	59 (89.4)	142 (92.8)				
CHEWS WRAPPERS	Present	38 (43.7)	23 (34.8)	61 (39.9)	0.27	1.45	0.75	2.81
	Absent	49 (56.3)	43 (65.2)	92 (60.1)				
NAIL BITING	Present	26 (29.9)	16 (24.2)	42 (27.5)	0.44	1.33	0.64	2.75
	Absent	61 (70.1)	50 (75.8)	111 (72.5)				
SUCKING FINGERS	Present	33 (37.9)	28 (42.4)	61 (39.9)	0.57	0.83	0.43	1.59
	Absent	54 (62.1)	38 (57.6)	92 (60.1)				
VENDORS	Present	42 (48.3)	32 (48.5)	74 (48.4)	0.98	0 .99	0.52	1.88
	Absent	45 (51.7)	34 (51.5)	79 (51.6)				
EATS POPSICLES	Present	78(89.7)	61 (92.4)	139 (90.8)	0.56	0.71	0.23	2.23
	Absent	9 (10.3)	5 (7.6)	14 (9.2)				
LICKS/BITES/CHEWS TOYS	Present	42 (48.3)	36 (54.5)	78 (51)	0.44	0.78	0.41	1.48
	Absent	45 (51.7)	30 (45.5)	75 (49)				
PLAYS WITH TYRES	Present	14 (16.1)	13 (19.7)	27 (17.6)	0.56	0.78	0.34	1.8
	Absent	73 (83.9)	53 (80.3)	126 (82.4)				
PLAYS WITH POTTERY	Present	2 (2.3)	1 (1.5)	3 (2)	0.73	1.53	0.14	17.24
	Absent	85 (97.7)	65 (98.5)	150(98)				
PLAYS WITH BATTERY	Present	5 (5.7)	6 (9.1)	11 (7.2)	0.43	0.61	0.18	2.09
	Absent	82(94.3)	60(90.9)	142 (92.8)				

Of the 153 participants, 22.9% had the habit of chewing or licking paint on the wall. Twenty eight percent (24/87) of the cases had the habit of chewing or licking paint on the wall as compares to 16.7 %(11/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 15.7% of the participants had the habit of eating mud. Seventeen percent (15/87) of the cases had the habit of eating mud as compared to 13.6% (9/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 38.6% had the habit of chewing doors/grills/window sills. Thirty-eight (33/87) of the cases had the habit of chewing doors/grills/window sills as compared to 39.4% (26/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 33.3% had the habit of chewing pencils or crayons. Twenty three percent (29/87) of the cases had the habit of chewing pencils or crayons as compared to 33.3% (22/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 7.2% had the habit of chewing chalk. Five percent (4/87) of the cases had the habit of chewing chalk as compared to 10.6% (7/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 39.9% had the habit of chewing toffee wrappers. Forty five percent (38/87) of the cases had the habit of chewing toffee wrappers as compared to 34.8% (23/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 27.5% had the habit of nail biting. Thirty percent (26/87) of the cases had the habit of nail biting as compared to 24.2% (16/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 39.9% had the habit of sucking on their fingers. Thirty eight percent (33/87) of the cases had the habit of sucking on their fingers as compared to 42.4% (28/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 48.4% had the habit of buying street food from vendors. Forty eight percent (42/87) of the cases had the habit of buying street food from vendors as compared to 48.5% (32/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 90.8% had the habit of eating popsicles. Ninety percent (78/87) of the cases had the habit of eating popsicles as compared to 92.4% (61/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 51% had the habit of chewing, biting or licking toys. Forty eight percent (42/87) of the cases had the habit of chewing, biting or licking toys as compared to 54.5%(36/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 17.6% had the habit of playing with tyres. Sixteen percent (14/87) of the cases had the habit of playing with tyres as compared to 19.7% (13/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 2% had the habit of playing with pottery. Two percent (2/87) of the cases had the habit of playing with pottery as compared to 1.5% (1/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 7.2% had the habit of playing with batteries. Six percent (5/87) of the cases had the habit of playing with batteries as compared to 9.1% (6/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Habit score:

The score was created by choosing ten habits which were common in our participants (lick/chews paint, eats mud, chews doors/grills/sills, chews, pencils/crayons, chews chalk, chews wrappers, nail biting, sucking fingers, vendors, licks/bites/chews toys).

The scores were then added to formulate a score from 0 to 10, where 0 is the end of the spectrum with no habits and 10 on the other end with all 10 habits. The control median of 4 was used as a cutoff to dichotomize the score to test association with elevated BLLs

TABLE 24: Habit score and elevated BLLs

No. of habits	Cases n (%)	Controls n (%)	Total n (%)
>=5	29 (33.3)	13 (19.7)	42 (27.5)
<5	58 (66.7)	53 (80.3)	111 (72.5)
Total	87	66	153

Among the 153 participants, 27.5% had more than>= 5 habits mentioned above. Of these, 33.3% (29/87) were cases and 19.7% (13/66) were controls. The remaining 72.5% had less than 4 habits. Even though the percentage of participants among cases with more than or equal to 5 habits was higher, there was no significant statistical association between those with more than 5 habits and elevated BLLs. (Chi sq. value=3.5, p-value= 0.06; OR=2.04, 95% CI: 0.96- 4.327)

7.6. Cosmetic use among participants and association with EBLLs

TABLE 25: Cosmetic use among cases and controls

VARIABLE		Cases n (%)	Controls n (%)	Total n (%)	Chi square P-value	OR	95 % CONFIDENCE INTERVAL	
							Lower	Upper
KAJAL	YES	80 (92)	57 (86.4)	137 (89.5)	0.26	1.8	0.64	5.13
	NO	7 (8)	9(13.6)	16 (10.5)				
SURMA	YES	2 (2.3)	1 (1.5)	3 (2)	0.73	1.53	0.14	17.24
	NO	85 (97.7)	65 (98.5)	150 (98)				
SINDOOR	YES	22 (25.3)	10 (15.2)	32 (20.9)	0.13	1.9	0.83	4.34
	NO	65 (74.7)	56 (84.8)	121 (79.1)				
FACE COSMETICS	YES	12 (13.8)	5 (7.6)	17 (11.1)	0.23	1.95	0.65	5.84
	NO	75 (86.2)	61 (92.4)	136 (88.9)				
LIP STICK	YES	13 (14.9)	10 (15.2)	23 (15)	0.97	0.98	0.4	2.41
	NO	74 (85.1)	56 (84.8)	130 (85)				
NAIL POLISH	YES	7 (8)	10 (15.2)	17 (11.1)	0.17	0.49	0.18	1.37
	NO	80 (92)	56 (84.8)	136 (88.9)				

Of the 153 participants, 89.5% had the habit of using kajal. Ninety percent (80/87) of the cases had the habit of using kajal as compared 86.4% (57/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs

Of the 153 participants, 2% had the habit of using surma. Two percent (2/87) of the cases had the habit of using surma as compared to1.5% (1/66) of the controls.

There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 20.9% had the habit of using sindoor. Twenty five percent (22/87) of the cases had the habit of using sindoor as controls 15.2% (10/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs

Of the 153 participants, 11.1% had the habit of using facial cosmetics (*saandhu/sandal tablets/tiruveni*). Fourteen percent (12/87) of the cases had the habit of using facial cosmetics as compared to 7.6% (5/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 15% had the habit of using lipstick. Fifteen percent (13/87) of the cases had the habit of using lipstick as compared to 15.2% (10/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Of the 153 participants, 11.1% had the habit of applying nail polish. Eight percent (7/87) of the cases had the habit of applying nail polish as compared to 15.2% (10/66) of the controls. There was no statistically significant difference between the presence of this habit and elevated BLLs.

Cosmetic score:

This score was made by adding 4 commonly used cosmetics (Kajal, Surma, Face cosmetics and Surma) and formulating a score from 0 to 4, where 0 is a participant who has not used any cosmetic item and 4 is the score given to a participant who has used all 4 cosmetic items. Using one or more cosmetics was used as the cut off to test for association between cosmetic use and elevated BLLs

TABLE 26. Cosmetic score and elevated BLLS

Items used	Cases n (%)	Controls n (%)	Total n (%)
1 or more	84 (96.6)	58 (87.9)	142 (92.8)
None	3 (3.4)	8 (12.1)	11 (7.2)
Total	87	66	153

Among the 153 participants, 92.8% used 1 or more cosmetics. Ninety seven percent (84/87) of the cases used 1 or more cosmetics as compared to 87.9% (58/66) of the controls. 11(7.2%) used none. Even though the percentage of participants among cases who used 1 or more cosmetic items was more, there was no statistically significant association between use of cosmetics and elevated blood levels. (Chi sq. value=4.2, p-value= 0.04* Fishers exact test =0.06; OR=3.86, 95% CI=0.98-15.18).

7.7. Hand wash practices before eating and association with EBLLs

TABLE 27: Hand washing before food and association with BLLs

HAND WASH	Cases n (%)	Controls n (%)	Total n (%)
NO	8 (9.2)	2 (3)	10 (6.5)
YES	79 (90.8)	64 (97)	143 (93.5)
	87	66	153

Among the 153 participants, 6.5% did not have the habit of washing their hands prior to eating food. Nine percent (8/87) of the cases did not have the habit of washing their hands prior to eating food as compared to 3%(2/66) of the controls. 143(93.5%) of participants did wash their hands prior to eating. Even though the percentage of cases who did not have the habit of washing hands prior to eating was higher, there was no statistically significant association between hand washing prior to eating and elevated BLLs. (Chi sq. value=2.34, p-value >0.05, Fishers exact test p =0.19; OR=3.24, 95% CI=0.67-15.80).

7.8. Smoking habits in the family and association with EBLLs

7.8.1. Smoker in the family:

TABLE 28: Presence of smoker in the family and association with BLLs:

Smoker in the family	Cases n (%)	Controls n (%)	Total n (%)
1 or more smokers	52 (60.5)	32 (48.5)	84 (55.3)
No smokers	34 (39.5)	34 (51.5)	68 (44.7)
Total	86	66	152

Among the 152 participants, 55.3% had 1 or more smokers in the family. Sixty one percent (52/86) of the cases had 1 or more smokers in the family as compared to 48.5% (32/66) of the controls. The remaining 68(44.7%) did not have any smokers at home. The presence of one or more smoker in the family was used to dichotomize the data for risk factor analysis. Even though the percentage of participants among cases having 1 or more smokers in the family was higher, there was no statistically significant association between smokers in the family and elevated BLLs. (Chi sq. value=2.17, p-value>0.05; OR=1.63, 95% CI=0.85-3.11).

7.8.2.Indoor smoking and association with EBLLs

TABLE 29: Indoor smoking and association with BLLs

Indoor smoking	Cases n (%)	Controls n (%)	Total n (%)
Yes	35 (40.2)	21 (31.8)	56 (36.6)
No	52 (59.8)	45 (68.2)	97 (63.4)
Total	87	66	153

Among the 153 participants, 36.6% had members who smoked indoors. Forty percent (35/87) of the cases had a indoor smoker in the family as compared to 31.8% (21/66) of the controls. Although the percentage of participants among cases with family members who smoked indoors was higher, there was no statistically significant association between indoor smoking and elevated BLLs. (Chi sq. value=1.14, p-value >0.05; OR=1.44, 95% CI=0.74-2.83).

7.9. Ingestion

7.9.1. Storage of drinking water:

TABLE 30: Drinking water stored in various containers associated with BLLs

Type of container	-	Cases n (%) N=87	Control n (%) N=66	Total n (%) N=153
Plastic	Yes	85 (97.7)	61(92.4)	146 (95.4)
	No	2 (2.3)	5 (7.6)	7 (4.6)
Brass	Yes	30 (34.5)	20 (30.3)	50 (32.7)
	No	57 (65.5)	46 (69.7)	103 (67.3)
Steel	Yes	14 (16.1)	12 (18.2)	26 (17)
	No	73 (83.9)	54 (81.8)	127 (83)

Drinking water stored in plastic containers:

Among the 153 participants, 95.4% households stored some of their drinking water in plastic containers. Ninety eight percent (85/87) of the cases households stored some of their drinking water as compared to 92.4% (61/66) of the controls. Even though the percentage of participant households among cases who stored part of their drinking water in plastic containers was higher, there was no statistically significant association between this practice and elevated BLLs. (Chi sq. value=2.39, p-value >0.05; **Fisher exact test: 0.20,** OR=3.48, 95% CI=0.65-18.55).

Drinking water stored in brass containers:

Among the 153 participants, 32.7% households stored some of their drinking water in brass containers. Thirty five percent (30/87) of the cases stored some of their drinking water in brass containers as compared to 30.3%(20/66) of the controls. Even though the percentage of participant households among cases who stored part of their drinking water in brass containers was higher, there was no statistically significant association between this practice and elevated BLLs. (Chi sq. value=0.30, p-value >0.05; OR=1.21, 95% CI=0.61-2.41).

Drinking water stored in steel containers:

Among the 153 participants, 17% households stored some of their drinking water in Steel containers. Sixteen percent (14/87) of the cases stored some of their drinking water in steel containers as compared to 18.2%(12/66) of the controls. There was no statistically significant association between this practice and elevated BLLs. (Chi sq. value=0.30, p-value >0.05; OR=1.21, 95% CI=0.61-

7.9.2. Having a piped water supply and association with EBLLs

TABLE 31: Piped water supply associated with EBLLs

Type of pipe	Cases n (%)	Controls n (%)	Total n (%)
Metal	75 (86.2)	53 (80.3)	128 (83.7)
Plastic	12 (13.8)	13 (19.7)	25 (16.3)
Total	87	66	153

Among the 153 participants, 83.7% participants received their water supply through iron pipes. Eighty six percent (75/87) of the cases received their water supply through iron pipes as compared 80.3% (53/66) of the controls. 25 (16.3%) received their water supply through plastic pipes and pipes made of other materials.

Metal pipes are established risk factor for elevated BLLs and was used to dichotomize for the Univariate analysis. Although the percentage of cases among the participants who received their water supply through iron pipes was higher, there was no statistically significant association between water delivery via iron pipes and elevated blood levels. (Chi sq. value=0.96, p-value >0.05; OR=1.53, 95% CI=0.65-3.62).

7.9.3.Use of plastics and melamine in the house:

TABLE 32:Use of plastics and melamine associated with EBLLs:

Plastic /Melamine	Cases n (%)	Controls n (%)	Total n (%)
Yes	13 (14.9)	10 (15.2)	23 (15)
No	74 (85.1)	56(84.8)	130 (85)
Total	87	66	153

Among the 153 participants, 15% used utensils made of plastic and melamine. Fifteen percent (13/87) of the cases used utensils made of plastic and melamine as compared to 15.2%(10/87) of the controls. 130(85%) participants did not use plastic or melamine at home. There was no statistically significant association between use of plastic or melamine utensils at home and elevated BLLs. (Chi sq. value=0.001, p-value>0.05; OR=0.98, 95% CI=0.40-2.41).

7.9.4. Use of brass in the house and association with EBLLs

TABLE 33:Use of brass in the house and association with BLLs:

Brass used at home	Cases n (%)	Controls n (%)	Total n (%)
Yes	50 (57.5)	34 (51.5)	84 (54.9)
No	37 (42.5)	32 (48.5)	69 (45.1)
Total	87	66	153

Among the 153 participants, 54.9% used utensils made of brass. Of these, 57.5% (50/87) were cases and 51.5 %(34/66) were controls. 69(45.1%) participants did not use brass at home. There was no statistically significant association between use of plastic or melamine utensils at home and elevated BLLs. (Chi sq. value=0.54, p-value >0.05; OR=1.27, 95% CI=0.67-2.42).

7.10.Environmental sources:

In each participant's house, multiple environmental samples were collected to assess environmental sources of lead. Drinking water, house dust, wall paint, door paint were samples collected if available in every house, while toys, kajal, cosmetics, grills paint samples were collected depending on the child's contact with it.

- Total samples collected: 693 samples
- Mean number of samples collected per house: 4.5

7.10.1. Drinking Water:

Among the study participants the mean (SD) and median (IQR) lead levels in the drinking water was 0.12 ppm (0.10) and 0.08 (0.04-0.19) ppm respectively, ranging from 0 to 0.43 ppm. The distribution of lead in drinking water has been explained in Table: 34

TABLE 34: Lead levels in the drinking water among the cases and controls

	Cases	Controls
Total samples	86	65
Mean (SD)	0.11(0.11)	0.12(0.09)
Median	0.076	0.09
Minimum	0	0
Maximum	0.43	0.35

TABLE 35: Lead in drinking water and association with elevated BLLs

Lead in drinking water (ppm)	Cases n (%)	Controls n (%)	Total n (%)
>=0.01	76(88.4)	62 (95.4)	138 (91.4)
<0.01	10 (11.6)	3 (4.6)	13 (8.6)
Total	86	65	151

Among the study participants 88% (76/86) of the cases had high lead levels in the drinking water, as compared to 95% (62/65) of the controls. The CDC cutoff for >=0.1 ppm was used as the cutoff for this risk factor analysis. This association was not statistically significant with elevated BLLs.(Chi sq. value=2.31, p-value>0.05; OR=0.37, 95% CI=0.1-1.40).

7.10.2. Wall paint:

Among the study participants 142 wall paint samples were collected from the households, the mean (SD) and median (IQR) lead levels in the wall paint was 3.96 ppm (16.6) and 0.59 (0.37-0.89) ppm respectively, ranging from 0 to 145 ppm. The distribution of lead in wall paint has been explained in TABLE: 36

TABLE 36: Lead levels in wall paint among cases and controls

	Cases	Controls
TOTAL	83	59
Mean (SD)	4.91(21)	2.62(6.5)
Median	0.58	0.61
Minimum	0.02	0
Maximum	145.08	36.6

TABLE 37: Lead levels in wall paint and association with elevated BLLs

Lead levels in wall paint	Cases n (%)	Controls n (%)	Total n (%)
High	37 (44.6)	30 (50.8)	67 (47.2)
Low	46 (55.4)	29 (49.2)	75 (52.8)
Total	83	59	142

Among the 142 wall paint samples 44.6%(37/83) of the cases had high lead levels in the wall paint as compared to 50.8% (30/59) of the controls. The median of the controls was used as the cutoff to test association with elevated BLLs. There was no statistically significant association between high lead levels in the wall paint and elevated BLLs.(Chi sq. value=0.54, p-value>0.05; OR=0.78, 95% CI=0.40-1.52).

7.10.3. Door paint:

Among the study participants 135 door paint samples were collected from the households, the mean (SD) and median (IQR) lead levels in the wall paint was 11.7 ppm (10.2) and 9.95 (4.13-16.57) ppm respectively, ranging from 0.05 to 63 ppm. The distribution of lead in door paint has been explained in Table: 38

TABLE 38: Lead levels in door paint among cases and controls

	Cases	Controls
Total	72	63
Mean (SD)	12.8 (11.6)	10.4 (8.3)
Median	9.985	8.99
Minimum	0.05	0.39
Maximum	63.26	39.05

TABLE 39: Lead levels in door paint and association with elevated BLLs

Lead levels in door paint	Cases n (%)	Controls n (%)	Total n (%)
High	40 (55.6)	32 (50.8)	72 (53.3)
Low	32 (44.4)	31 (49.2)	63 (46.7)
Total	72	63	135

Among the 135 door paint samples analyzed, 55.6% (40/72) of the cases had high lead levels in the door paint as compared to 50.8% (32/63) of the controls. The median of the controls was used as the cutoff to test association with elevated BLLs. There was no statistically significant association between high lead levels in the wall paint and elevated BLLs.(Chi sq. value=0.306, p-value>0.05; OR=1.2, 95% CI=0.62-2.4).

7.10.4. House dust:

Among the study participants 143 house dust samples were collected from the households, the mean (SD) and median (IQR) lead levels in the house dust was 0.69 ppm (0.97) and 0.51(0.28-0.77) ppm respectively, ranging from 0 to 9.73 ppm. The distribution of lead in house dust has been explained in Table: 40

TABLE 40: Lead levels in house dust among cases and controls

	Cases	Controls
Total	82	61
Mean (SD)	0.69(1.14)	0.68(0.69)
Median	0.505	0.52
Minimum	0	0
Maximum	9.73	4.52

TABLE 41: Lead levels in house dust and association with elevated BLLs

Lead levels in house dust	Cases n (%)	Controls n (%)	Total n (%)
High	39 (47.6)	31 (50.8)	70 (49)
Low	43(52.4)	30 (49.2)	73 (51)
Total	82	61	143

Among the 143 house dust samples analyzed, 47.6% (39/82) of the cases had high lead levels in the house dust as compared to 50.8% (31/61) of the controls. The median of the controls was used as the cutoff to test association with elevated BLLs. There was no statistically significant association between high lead levels in the wall paint and elevated BLLs. (Chi sq. value=0.15, p-value>0.05; OR=0.88, 95% CI=0.45-1.70).

7.10.5. Toys:

Among the study participants 36 toys which the children used were collected from the households, the mean (SD) and median (IQR) lead levels in the house dust was 0.35 ppm (0.35) and 0.25 (0.08-0.25) ppm respectively, ranging from 0 to 1.65 ppm. The distribution of lead in toys has been explained in Table: 42

TABLE 42: Lead levels in Toys among cases and controls

	Cases	Controls
Total	21	15
Mean (SD)	0.39 (0.38)	0.29 (0.3)
Median	0.3	0.22
Minimum	0	0
Maximum	1.65	1

TABLE 43: Lead levels in toys and association with elevated BLLs

Lead levels in toys	**Cases n (%)**	**Controls n (%)**	**Total n (%)**
High	14(66.7)	8 (53.3)	22 (61.1)
Low	7(33.3)	7 (46.7)	14 (38.9)
Total	21	15	36

Among the 36 toy samples analyzed, 66.7% (14/21) of the cases had high lead levels in the toys as compared to 53.3% (8/15) of the controls. The median of the controls was used as the cutoff to test association with elevated BLLs. There was no statistically significant association between high lead levels in the wall paint and elevated BLLs.(Chi sq. value=0.66, p-value>0.05; OR=1.75, 95% CI=0.45-6.83).

7.10.6. Kajal:

Among the study participants 121 kajal which the children used were collected from the households, the mean (SD) and median (IQR) lead levels in the house dust was 6.55 ppm (8.14) and 4.57 (4.57-4.57) ppm respectively, ranging from 0 to 1.65 ppm. The distribution of lead in toys has been explained in Table: 44.

TABLE 44: Lead levels in Kajal among cases and controls

	Cases	Controls
Total	48	73
Mean (SD)	6.39 (8.5)	6.66(7.95)
Median	4.57	4.57
Minimum	0.14	0.07
Maximum	33.88	33.88

TABLE 45: Lead levels in kajal and association with elevated BLLs

Lead levels in kajal	**Cases n (%)**	**Controls n (%)**	**Total n (%)**
High	69 (94.5)	41 (85.4)	110 (90.9)
Low	4 (5.5)	7 (14.6)	11 (9.1)
Total	73	48	121

Among the 121 kajal samples analyzed, 94.5% (69/73) of the cases had high lead levels in the kajal used as compared to 85.4% (41/48) of the controls. There was no statistically significant association between high lead levels in the wall paint and elevated BLLs. (Chi sq. value=2.95, p-value>0.05; OR=2.95, 95% CI=0.81-10.68).

7.10.7.Comparing Mean values of lead among different environmental samples tested between cases and controls

The Independent t-test was done to see if there was any significant difference in the mean values between the cases and controls cases in each environmental sample. Even though the mean lead levels in wall paint door paint and kajal were higher there was no statistically significance between cases and controls.

TABLE 46. Independent t-test of the environmental samples

Sample	Case /Control	Total	Mean	Std. Deviation	Mean Difference	t-test Value	Significance
Water	Case	86	0.11	0.11	-0.01	-0.53	0.6
	Control	65	0.12	0.09			
Wall Paint	Case	83	4.91	21.07	2.29	0.93	0.36
	Control	59	2.62	6.53			
Door paint	Case	72	12.81	11.58	2.37	1.38	0.17
	Control	63	10.44	8.32			
House dust	Case	82	0.69	1.14	0.01	0.06	0.95
	Control	61	0.68	0.69			
Grill window paint	Case	7	11.06	14.22	-4.23	-0.59	0.57
	Control	8	15.29	13.41			
Kajal	Case	6	4.66	10.93	4.37	0.98	0.37
	Control	6	0.3	0.15			
Sindoor	Case	8	0.35	0.16	-1.72	-0.96	0.38
	Control	7	2.08	4.76			
Toys	Case	21	0.39	0.38	0.09	0.83	0.42
	Control	15	0.29	0.3			

7.10.8. Correlation between lead in environmental samples and elevated blood lead levels among the study participants

Correlation between BLLs and each environmental sample was tested to identify any significant correlation between the two but there was no significant correlation noted. The details of correlation are presented in Table: 47

TABLE 47: Correlation between BLLs and environmental samples

Sample	Pearson's correlation coeff.	P value
Water	0.22	0.18
Wall paint	0.02	0.86
Door paint	0.17	0.05
House dust	0.02	0.82
Grill window	-0.33	0.24
Kajal	-0.02	0.957
Toys	0.05	0.77

7.11. Summary table of univariate analysis:

The summary of univariate risk factor analyses for important socio-demographic and household environment and elevated BLLs among study participants is provided in Table 48 below. Painting the house once or more number of times in the last 5 years and age of the house >=10 years emerged as significant risk factors for EBLLs

TABLE 48: Summary of univariate analysis

Variable	Risk factor	Cases n (%) N=87	Control n (%) N=66	Total n (%)	Chi sq p-value	OR	Lower	Upper
Gender	Male	37 (42.5)	35(53)	72(47.1)	0.197	0.66	0.34	1.25
EBF	<6 months	49 (56.3)	30 (45.5)	79 (51.6)	0.183	1.55	0.81	2.95
Religion	Muslim	22 (25.3)	12 (18.2)	34 (22.2)	1.1	1.52	0.69	3.36
Birth weight	Low birth weight	9 (10.8)	15 (23.1)	24 (16.2)	0.045	0.41	0.17	1
Number of members in the household	>=6	52 (59.8)	35 (53)	87 (56.9)	0.404	1.32	0.69	2.51
Number of children living in each household	>=3	40 (46)	35 (53.8)	75 (49.3)	0.337	0.73	0.38	1.39
Child death in the family	Yes	10 (11.5)	4 (6.1)	14 (9.2)	0.25(0.27)	2.01	0.6	6.73
High risk occupation	Yes	19(23.2)	13(21.7)	32 (22.5)	0.832	1.09	0.49	2.43
Number of people per room	>=3	52(59.8)	37 (56.1)	89 (58.2)	0.645	1.16	0.61	2.23
Type of roof	Asbestos/tin	23 (26.4)	12 (18.2)	35 (22.9)	0.229	1.62	0.74	3.55
Type of floor	Earth/mud/clay	7(8)	2 (3)	9 (5.9)	0.28(0.47)	2.8	0.56	13.95
Type of wall	Mud	19 (21.8)	8 (12.1)	27 (17.6)	0.118	2.03	0.83	4.97
Type of paint	Emulsion/enamel	15 (17.4)	13 (21.3)	28 (19)	0.556	0.78	0.341	1.786
Times painted last 5 years	>=1	80 (93)	49 (80.3)	129 (87.8)	0.021*	3.27	1.5	9.26
Age of the house in years	>=10	70 (80.5)	37 (56.1)	107 (69.9)	0.001*	3.23	1.57	6.63
Factory near the house	Yes	6 (6.9)	6 (9.1)	12 (7.8)	0.617	0.74	0.22	2.41
Times swept per day	>=4	64 (73.6)	40 (60.6)	104 (68)	0.09	1.81	0.91	3.52
House washed per week	1 to2	57 (65.5)	36 (54.5)	93 (60.8)	0.169	1.58	0.82	3.05
No. Of habits	>=5	29 (33.3)	13 (19.7)	42 (27.5)	0.061	2.04	0.96	4.33
Cosmetic Items used	1 or more	84 (96.6)	58 (87.9)	142 (92.8)	0.04*	3.86	0.98	15.18

Variable	Risk factor	Cases n (%) N=87	Control n (%) N=66	Total n (%)	Chi sq p-value	OR	Lower	Upper
Hand wash before eating	No	8 (9.2)	2 (3)	10 (6.5)	0.13(0.19)	3.24	0.67	15.8
Smoker in the family	1 or more smokers	52 (60.5)	32 (48.5)	84 (55.3)	0.141	1.63	0.85	3.11
Indoor smoking	Yes	35 (40.2)	21 (31.8)	56 (36.6)	0.285	1.44	0.74	2.83
Drinking water stored in plastic containers	Yes	85 (97.7)	61(92.4)	146 (95.4)	0.122	3.48	0.65	18.55
Drinking water stored in brass containers	Yes	30 (34.5)	20 (30.3)	50 (32.7)	0.585	1.21	0.61	2.41
Drinking water stored in steel containers	Yes	14 (16.1)	12 (18.2)	26 (17)	0.733	0.863	0.37	2.014
Piped water supply	Metal	75 (86.2)	53 (80.3)	128 (83.7)	0.328	1.53	0.65	3.62
Use of plastics and melamine	Yes	13 (14.9)	10 (15.2)	23 (15)	0.971	0.98	0.4	2.41
Use of brass in the house	Yes	50 (57.5)	34 (51.5)	84 (54.9)	0.463	1.27	0.67	2.42
Lead in drinking water (ppm)	>=0.01	76(88.4)	62 (95.4)	138 (91.4)	0.128	0.37	0.1	1.4
Lead levels in wall paint	High	37 (44.6)	30 (50.8)	67 (47.2)	0.46	0.78	0.4	1.52
Lead levels in door paint	High	40 (55.6)	32 (50.8)	72 (53.3)	0.58	1.2	0.62	2.39
Lead levels in house dust	High	39 (47.6)	31 (50.8)	70 (49)	0.7	0.88	0.45	1.7
Lead levels in toys	High	14(66.7)	8 (53.3)	22 (61.1)	0.418	1.75	0.45	6.83
Lead levels in kajal	High	69 (94.5)	41 (85.4)	110 (90.9)	0.088	2.95	0.81	10.68

7.12.Multivariate analysis by logistic regression

To adjust for potential confounders, selected variables which were strongly established risk factors from this and other studies or chi square p- value up to 0.1 were chosen from the univariate analyses for multivariate analysis. A logistic regression analysis was performed to study the risk factors for elevated BLLs among children at 24 months after adjusting for potential confounders. The following variables were used as exposure variables: Exclusive breast feeding <6 months, low birth weight, Child death in the family, number of members in the family (>=6), Fathers high risk occupation, asbestos or tin roof, House age >=10years, house painted >=1 once in the last 5 years, house swept>=4 times per day, house washed <3 times per week, >= 5 habit of the child, cosmetic use of 1 or more items, no hand wash prior to eating, metal piped water supply, drinking water stored in plastic containers, Brass use at home, presence of one or more smokers in the family. The models statistics are presented in Table: 49.

TABLE NO 49: Logistic regression model for factors associated with elevated BLLs

Variable	OR (unadjusted)	95% C.I (unadjusted)		Chi square p-value	OR (Adjusted)	95% C.I (adjusted OR)	
		Lower	Upper			Lower	Upper
Exclusive breast feeding(<6 mths)	1.55	0.81	2.95	0.39	1.88	0.45	7.94
Low birth weight	0.41	0.17	1	0.09	0.35	0.1	1.19
Child death in the family >=6	2.01	0.6	6.73	0.11	3.96	0.73	21.58
Number of members in the household	1.32	0.69	2.51	0.24	1.69	0.7	4.08
Father high risk occupation	1.09	0.49	2.43	0.21	2	0.69	5.83
Asbestos/tin roof	1.62	0.74	3.55	0.21	2.14	0.654	6.999
House age >=10years	3.23	1.57	6.63	<0.001*	6.53	2.43	17.58
House painted in the last 5 years (>=1)	3.27	1.5	9.26	0.004*	7.05	1.84	26.99
Times swept per day (>=4)	1.81	0.91	3.52	0.34	0.64	0.25	1.6
House washed weekly (<3times)	1.58	0.82	3.05	0.65	1.22	0.52	2.83
Habit score (>=5)	2.04	0.96	4.33	0.045*	2.93	1.03	8.37
Cosmetic use (>=1 item)	3.86	0.98	15.18	0.64	1.56	0.25	9.86
No hand wash prior to eating	3.24	0.67	15.8	0.13	0.23	0.04	1.52
Metal piped water supply	1.53	0.65	3.62	0.39	1.63	0.54	4.89
Drinking water stored in plastic	3.48	0.65	18.55	0.046*	8.06	1.04	62.27
Brass used at home	1.27	0.67	2.42	0.07	2.32	0.95	5.68
Smoker In The Family	1.63	0.85	3.11	0.66	1.22	0.5	2.99

In this regression model House painted last more than or equal to once in the last5 years [Adjusted OR 7.05 (95%CI 1.84-26.99)], Age of the house more than or equal to 10 years [Adjusted OR 6.53 (95%CI 2.43-17.58)], Drinking water stored in plastic containers [Adjusted OR 8.06 (95%CI 1.04-62.27)] and a habit score more than or equal to 5 [Adjusted OR 2.93 (95%CI 1.03-8.37] were associated with elevated BLLs.

8 Discussion

In this community based case control study, the main objectives were to study the risk factors for elevated blood lead levels among pre-school children residing urban slums of Vellore and assess the lead levels in their living environment. The mean (SD) BLLs of the 224 children from the primary MAL-ED study was 12.6(8.5) mcg/dl, the median was 10.5 mcg/dl and the BLLs ranged from 1.5 to 66.8mcg/dl. Nearly 97% (166/224) of the children from the cohort had elevated BLLs based on the recent CDC recommended value of 5mcg/dl. Using the earlier cut-off value of 10mcg/dl, more than half (132/224) of the children had elevated BLLs at 24 months of age, thus indicating that lead poisoning is a major public health problem among children living in these communities.

Among the study participants, 3 (2%) had BLLs of 0 to 4.9 mcg/dl, 63 (41.2%) participants had BLLs of 5 to 9.9 mcg/dl, 55 (35.9%) participants had BLLs of 10 to 14.9 mcg/dl and 32 (20.9%) participants had BLLs more than 15 mcg/dl

In this case control study using the cut off value of 10mcg/dl, 87 cases and 66 controls were studied to assess the risk factors for elevated blood lead levels at 24 months of age and also to estimate the lead levels in their living environment. The means (SD) of BLLs were 16.6 (9.4) and 7.4 (1.9) mcg/dl among cases and controls respectively. It has been well documented that the burden of lead poisoning varies between countries and also between different regions within a country. Limited information is available from India on the burden of blood lead levels among children with individual studies addressing the problem among school going children. The proportion of children with elevated BLLs in this region was higher than those reported from other parts of India (12 to 38%). (20,22,73) Most of the other studies from India were not conducted in slums, so the exposure to lead may be higher in a slum as compare to other residential areas.

Socio-demographic correlates for lead poisoning

Only a few studies are available from India looking at factors that might correlate with BLLs among children. Constraints with information of lead measurements and study designs have been an issue in India. At the individual or family level, the significant predictors associated with elevated BLLs among children include educational status of the parents or the primary caregiver, age of the children; lower SES, total number of children borne to the mother and nutritional status in the developing countries.

In this study, even though there were more females among the cases than the controls (58% vs.47%), gender of the child was not significantly associated with EBLLs.

In the slums studied, majority of the study families belonged to the upper lower class (58%) followed lower middle class (33%) and upper middle class (9%) and commonly seen occupations among the fathers were daily wage work as load lifters in the local vegetable market in Vellore.

Majority of the participants were living in extended families (46%) followed by nuclear (40%) and joint families (14%). The mean (SD) number of people in each family was 6.3 (2.1), ranging from 2 to 13 people. Children from families having 6 or more members had 32% excess risk of having elevated BLLs and this finding was not statistically significant. Similarly there was no statistically significant association noted between number of children in the family and EBLLs in this study. With an increase in family size and overcrowding, there is a potential for increased exposure to lead from other family members.

Some of the studies have identified any past child death in the family as a potential risk factor for lead poisoning. Death of any child in the family can be a surrogate indicator for multiple other factors measuring vulnerability to infections, malnutrition, poorer socio-economic status, inadequate child rearing practices, health seeking behavior of the family etc. In this study while participants from families with any past child death had double the risk of having elevated BLLs on univariate analysis which further increased to 4 times after adjusting for other confounders in multivariate logistic regression analysis, this association however was not statistically significant.

Parental occupation and lead poisoning:

Parents could be major contributors for lead exposure among. It has been demonstrated that people working in occupations involving lead carry lead dust on their clothes, hair and nails. Many studies have shown that there is a significant association between such occupations and EBLLs among children as majority of parents come back home and spend time with children by playing or carrying them, without changing out of work clothes, bathing or even washing hands.(3) In this study, a total of 32 out of the 142 working fathers, worked in high-risk occupations where lead could be used. Among the cases, 23% (19/82) fathers of children with elevated BLLs were involved in high-risk occupations and among the controls 22 %(13/60) worked in High risk occupations. There was no statistically significant association between high-risk occupations and EBLLs.

Housing characteristics:

Majority of the study houses were overcrowded with mean (SD) number of people per room being 3 (1.5), ranging from 1 to 9 people per room. This also means that there is increased exposure more so if other family members involved in high risk occupations. In this study we did not find overcrowding to be significantly associated with lead poisoning.

It is generally considered that concrete roofs are safer and more protective against lead poisoning as compared to other make shift, painted, more porous and less durable roofs. Majority of the participants (50%) lived in houses with Concrete roofs, while the others lived in houses with tiled, thatched, tin or asbestos roofs. Even though higher proportion of cases (26.4 Vs. 18.2%) living under other roofs like asbestos and tin as compared to controls, this was not statistically significant.

In a similar study done on the same participants houses with earth /mud /clay floors were significantly associated with 15 month EBLLs.(4) But it association was not consistent at 24 months as noted in this study.

Lead in paint has always been a matter of concern, especially because paint is readily available in the child's immediate surroundings. As the paint starts aging and peeling of surfaces such as walls, windows, doors and grills, children are more prone to eat the lead containing flaking paint or even ingest dust rich in lead contributed by the flaking paint. The phenomenon is often seen in older paints which were used due to lack of legislation regarding lead concentrations in paint nearly 20 years back in India. Age of the house is thus an important risk factor associated with EBLLs. This study also showed similar results that the odds of a participant with EBLLs was 3 times more likely if he stayed in a house >= 15 years of age. This is likely to because the older houses still have layers of older paint under newer ones, slowly contributing to the dust in the child's environment, which is ingested by the child due to increased hand to mouth activities. In most houses even if a newer layer of paint has to be painted, a few layers of older paint is always scraped of and sand papered to make the base smooth for the newer paint to adhere better. This process can also rack up lead laden dust contributing to EBLLs. Majority of the houses (88%) were re-painted within the last five years and the association was statistically significant in the univariate analysis and also after adjusting for potential confounders in the multivariate analysis.

Habits of the children and association with elevated blood lead levels:

Preschool children often have increased hand to mouth activities and habits, which contribute to the ingestion of lead. Licking, chewing, eating paint from walls and grills were significantly associated with EBLLs in some studies, especially in children due to the characteristic sweet taste of lead.(40) In this study there was no significant association between each individual habit and EBLLs even though the proportions were higher among cases. When child behavior with respect to having multiple habits was considered, it was noted that children having 5 or more of the above listed habits were significantly had 2.8 times higher odds of having EBLLs possibly indicating multiple sources of contamination and the effect of cumulative exposure on them; this finding remained significant even after adjusting for various other confounders in the study.

Many studies have shown increased lead concentrations in the paint used in pencils, crayons, toys and even chocolate wrappers and children biting and chewing these items were associated with EBLLs, although it was not significant in this study.(3)

Nail biting, finger sucking and eating mud were also not significantly associated with EBLLs.

Usage of Cosmetics and their association with elevated blood lead levels:

Lead imparts luster and shimmer when added to cosmetics and thus is widely used due to its availably and cost. A study done in Saudi Arabiahas reported cosmetic use as a risk factor for elevated BLLs.(13) In this study, kajal was applied to vast majority (89%) of the participants. Regular application of one or more cosmetic item on children among kajal, surma, sindoor and face cosmetics) was significantly associated with EBLLs in the univariate analysis, which however was not significant in the adjusted multivariate analysis.

Ingestion as a method of lead exposure

In this study, children living in houses where water was supplied through metal pipes, and houses with brass utensils had higher risk of having elevated blood lead levels although this finding was not statistically significant. Children from families where drinking water was stored in plastic containers had significantly higher blood lead levels (OR = 8) after adjusting for other risk factors in the study. Water can get contaminated with lead in areas where lead pipes are primarily used for supply and also lead is mixed in various substances to improve the tensile strength of that substance, especially in big plastic containers and poly vinyl chloride pipes.

Other risk factors:

Other risk factors of EBLLs like Exclusive breast feeding >=6 months, hand washing before eating, presence smokers in the family and indoor smoking were also analysed and were not found to be significantly associated with EBLLs among children at 24 months in this study.

Environmental sources:

The second objective of the study was to estimate the lead levels in environmental sources and test their association with lead poisoning among children.

The Environmental Protection Agency (EPA), USA has recommended cutoff value for lead in drinking water as 15mcg/l, which is equal to 0.015ppm of lead in water. Using this value as the cutoff in the drinking water in this study, 91.4 % of the drinking water samples collected from homes of participants had levels exceeding 0.015ppm. This finding is very high and alarming when compared to other studies around the world. Although there was no statistically significant association between lead levels exceeding 0.01ppm in drinking water and EBLLs, it is a matter of concern knowing that majority of the preschool children in this area are exposed to such high levels of lead in water especially with the goal of zero tolerance lead in drinking water as a recommended goal in USA. This part of the city still continues to have older lead pipes in the main water distribution network, which could contaminate drinking water. Also since most of the families store drinking water in plastic containers, which are bright coloured plastic pots (kodams), the material and the paint of these could also contribute to the contamination process.

Paints are a major source of lead in the environment. The EPA has set a standard of 600 ppm of lead as the upper limit in paints. In India, though there are no regulatory limits, a voluntary standard of 1000 ppm is recommended for lead in paints. A recent study looking at the lead composition in different types of paints sold by major companies has shown that even though plastic or water based paints had lesser concentrations of lead, 84% of the enamel based paints in India had concentrations above 1000 ppm and 61% had more than 5000 ppm.(2) In this study, although the mean paint concentrations of lead in paint scraped from walls, windows, grills, gates and doors were higher in cases as compared to controls the difference was not statically significant. The standard way of estimation of lead in paints is by using raw paint solutions or using portable X-ray fluorescent spectrometry on painted surfaces. Although this study employed another standard recommended technique FAAS, obtaining the samples from walls, door and windows by scraping may have influenced the results.

Dust and soil contamination in painted houses are important routes of exposure among children in addition to them eating chips of paint.(2) The mean levels of lead in house dusts were similar among both cases and controls in this study. In slums where houses are generally in close proximity to one another in a small geographic area and where families with poorer sections of the society live together, one could expect similar contamination levels in the living environments.

Lead may be found in the paint on the toys or in the plastics which they are made of Studies have shown that people perceive that toys manufactured in the developing countries are unsafe and were willing not to use them. In the year 2007, more than 675,000 toys manufactured by a leading company were recalled in the United States as the surface paints on these toys were found to contain excessive lead.(50;86) Some of the products manufactured in China which were recalled in the United States due to high lead levels include children's necklaces and bracelets, chalk and pet foods apart from toys.(87) A study from India looking at lead and cadmium in soft plastic toys from three metropolitan cities revealed that all the toys examined showed the presence of lead and cadmium in them, while 20% of the toys had excess concentrations of lead.(57) In this study lead levels in toys and cosmetics were not significantly associated with EBLLs among the study participants.

9. Limitations

- One of the main setbacks of this study could be "recall bias" due to the time lag of nearly 6 months to 2 years in some participants from blood collection at 24 months to the time of interview. During this period many habits, cosmetic use and other risk factors of the child could have also changed.
- Many parents had discarded the older toys used by the participant at the time of blood collection.
- Many participants shifted houses or moved out of the area, we could not obtain equal number of cases and controls.
- Lead in house dust was estimated from floor sweepings as compared to the standard vacuum collections or wet wipe sampling for lead estimation.
- Other intrinsic factors like anemia, calcium deficiency and nutritional status, which are also important, factors contributing to EBLLs were not taken into consideration in this study.

10. Summary and conclusion

Elevated BLLs is a major public health problem among preschool children living in urban slums of Vellore.

- Blood lead levels (Primary study)
    - 46.8% of the participants had BLLs >= 10 mcg/dL.
    - 98% of the participants had BLLs >=5 mcg/dL.
- Risk factors for elevated blood lead levels
    - Univariate analysis:
        i. House being painted at least once in the last 5 years.
        ii. Age of the house >=10 years.
        iii. Use of at least 1 cosmetic item (bordering on significance).
    - Multivariate analysis:
        i. House being painted at least once in the last 5 years.
        ii. Age of the house >=10 years.
        iii. Habit score of >= 5 habits per child.
        iv. Drinking water stored in plastic container.
- Environmental lead levels
    - 91.4% of the drinking water samples had Lead levels more than the CDC recommendation for lead in drinking water.
    - Although the mean lead levels in wall paint, door paint and toys were higher, there was no significant association with elevated BLLs.

More research focusing on environmental sources of EBLLs is needed to understand the exposure pathways in greater detail and to establish causality in this region.

11 Recommendations

1. Houses which more than 10 years of age should conform to the minimal housing standards in terms of paints previously used.
2. The households with children in them have to be educated about the risks and hazards of painting and the painting process and its detrimental effects on children.
3. Drinking water storage practices must be studied more in detail to established concrete evidence in associations between drinking water and EBLLs.
4. Health Education to parent regarding –child behavior, habits and use of quality cosmetics is necessary to decrease lead exposure.
5. Education to the parent about work exposure and hygiene after work, hand washing, smoking and indoor smoking also needs to be stressed on.

12 Bibliography

1. World Health Organization. Lead in Drinking-water. Geneva: WHO; 2011. 19 p.
2. Kumar A. A Brush with Toxics-An investigation into lead in household paints in India. New Delhi: Toxics Link; 2007. 36 p.
3. World Health Organization. Childhood Lead Poisoning. Geneva: WHO; 2010.
4. Mohan VR, Sharma S, Ramanujam K, Babji S, Koshy B, Bondu JD, et al. Effects of Elevated Blood Lead Levels in Preschool Children in Urban Vellore. Indian Paediatrics. 2014;51:621-5.
5. Eisinger J. Lead and wine: Eberhard Gockel and the colica Pictonum. Medical History. 1982 (26):279–302.
6. Franklin B. Letter on lead poisoning to Benjamin Vaughan. 1786.
7. Planches TD. Traite des maladies de plomb ou saturnines. Paris 1839.
8. Thackrah C. The effects of arts, trades, and professions, and of civic states and habits of living, on health and longevity: with suggestions for the removal of many of the agents which produce disease, and shorten the duration of life. In: Longman, editor. 2 ed. London1832.
9. Gibson JL. A plea for painted railings and painted walls of rooms as the source of lead poisoning amongst Queensland children. 1904. Public health reports. 2005 May-Jun;120(3):301-4..
10. Michaelowa A. Phasing out in gasoline — how developing countries can learn from the experiences of the industrialized world. In: . World development aid and joint venture finance 1997:268–72.
11. Singh AK, Singh M. Lead decline in the Indian environment resulting from the petrol-lead phase-out programme. Science of the Total Environment 2006 (368):686–94.
12. Rahbar M, White F, Agboatwalla M, Hozhabri S, Luby S. Factors associated with elevated blood concentration in children in Karachi, Pakistan. Bull World Health Organ 2002;80:769–78.
13. Zolaly M, Hanafi M, Shawky N, Harbi KE, Mohamadin. Association between blood lead levels and environmental exposure among Saudi school children in certain districts of Al-Madinah. Am Int J Gen Med. 2012;5:355-64.
14. Albalak, Noonan R, Buchanan G, Flanders S, Gotway-Crawford WD, C Kim, et al. Blood lead levels and risk factors for lead poisoning among children in Jakarta, Indonesia. The Science of the total environment. 2003 Jan 1;301(1-3):75-85.
15. Riddell TJ, Solon O, Quimbo SA, Tan CMC, Butrick E, Peabody JW. Elevated blood-lead levels among children living in the rural Philippines. Geneva: WHO; 2007.
16. Agency for Toxic Substances and Disease Registry. Toxicological Profile for lead. Atlanta: US Department of Health and Human Services; 1999.

17. World Health Organization. Global burden of disease: 2004 update. Switzerland: WHO; 2004.

18. Lovei M. Phasing out lead from gasoline: worldwide experience and policy implications. Washington DC: 1996.
19. Jain NB, Hu H. Childhood Correlates of Blood Lead Levels in Mumbai and Delhi. Environ Health Perspect. 2006;114 (3).
20. Landrigan P. The worldwide problem of lead in Petrol. Bull World Health Organ 2002;80(768).

21. Rojas-López. Use of lead-glazed ceramics is the main factor associated to high lead in blood levels in two Mexican rural communities. J Toxicol Environ Health. 1994;42(1):45-52.
22. Mostafa G, El-Shahawi H, Mokhtar A. Blood lead levels in Egyptian children from high and low lead-polluted areas: impact on cognitive function. Acta Neurol Scand. 2009;120:30–7.
23. Ghose M. Controlling of motor vehicle emissions for a sustainable city. TIDE 2002;1(2):273-82.
24. Singh N, Yunus M, Srivastava K, Singh S, Pandey V, Mishra J, et al. Monitoring of auto exhaust pollution by roadside plants. Environ Monit Asses. 1995;34:13-25.
25. Kalra V, Sahu JK, Bedi P, Pandey RM. Blood Lead Levels among School Children after Phasing-Out of Leaded Petrol in Delhi, India. Indian J Pediatr. 2013;10.
26. The George Foundation. A study of lead poisoning in major Indian cities. Proceedings of the International Conference on lead poisoning prevention and treatment. Bangalore: The George Foundation; 1999 February 8–10, 1999. 79–85 p.
27. Jain N, Hu H. Childhood correlates of blood lead levels in Mumbai and Delhi. Environmental health perspectives. 2006;114:466-70.
28. Bellinger D, Hu H, Kalaniti K, Thomas N, Rajan P, Sambandam S, et al. A pilot study of blood lead levels and neurobehavioral function in children living in chennai, India. Int J Occup Environ Health. 2005;11(2):138–43.
29. Nichani V, WI L, Smith M, Noonan G, Kulkarni M, Kodavor M, et al. Blood lead levels in children after phase-out of leaded gasoline in Bombay, India. The Science of the total environment. 2006 2006 Jun 15;363(1-3):95-106.
30. Kalra V, Chitralekha KT, Dua T, Pandey RM, Gupta Y. Blood Lead Levels and Risk Factors for Lead Toxicity in Children from Schools and an Urban Slum in Delhi . Journal of tropical pediatrics.49(2).
31. Pirkle J, Brody D, Gunter E. The decline in blood lead levels in the United States. The national health and nutrition examination surveys. . JAMA : the journal of the American Medical Association. 1994;272:284-91.
32. Tong S, Schirnding V, Prapamontol T. Environmental lead exposure: a public health problem of global dimensions. Bull World Health Organ 2000;78:1068–77.
33. Lanphear B, Byrd R, Auinger P, Schaffer S. Community characteristics associated with elevated blood lead levels in children. Pediatrics. 1998;101:264-71.
34. Staffan S, Ingvar A, Bergdahl. LEAD. Handbook on the Toxicology of Metals 2007.
35. World Health Organization. Air quality guidelines for Europe. Copenhagen:WHO; 2000.
36. Patel KS, Ambade B, Sharma S, Sahu D, Jaiswal NK, Gupta S, et al. Lead Environmental Pollution in Central India. New Trends in Technologies. 2010;978:62-6.
37. Guidelines for drinking-water quality: incorporating 1st and 2nd addenda. 3 ed. Geneva: World Health Organization 2008.
38. Clark C, Rampal K, Thuppil V, Roda S, PMSuccop, Menrath W. Lead levels in new enamel household paints from Asia, Africa and South America. Environ Res J. 2009;109(7):930–6.
39. Ban of lead-containing paint and certain consumer products bearing lead-containing paint: Consumer Product Safety Commission; 1977.
40. Vishwanath P, Devegowda D, Prashant A, Nayak N, D'souza V, Venkatesh T. Environmental lead levels in a coastal city of India: the lead burden continues. Indian J Med Sci. 2012;66(11-12):260–6.

41. RS Tsutsui , Schalkwyk JV, Spriggs D. Lead poisoning from Ayurvedic medicines. The New Zealand medical journal. 2013;126(1374):80-3.
42. Baker EL. Lead poisoning in children of lead workers: home contamination with industrial dust. N Engl J Med. 1977;296(5):260-1.
43. Chisolm J. Fouling one's own nest. Pediatrics. 1978;62:614-7.
44. Markowitz M, Rosen J, Clemente. Clinician follow-up of children screened for lead poisoning. Am J Public Health. 1999 Jul;89(7):1088-90.
45. Lidsky T, Schneider J. Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain. 2003;126(1):5-19.
46. Gulson B. Mobilization of lead from human bone tissue during pregnancy and lactation – a summary of long-term research. Sci Total Environ. 2003;303(1-2):79-104.
47. Ettinger A. Influence of maternal bone lead burden and calcium intake on levels of lead in breast milk over the course of lactation. Am J Epidemiol. 2006;163(1):48–56.
48. Leung A. Heavy metals concentrations of surface dust from e-waste recycling and its human health implications in southeast China. Int J Environ Sci Technol. 2008;42:2674–80.
49. Zheng L. Blood lead and cadmium levels and relevant factors among children from an e-waste recycling town in China. Environ Res J. 2008;108:15-20.
50. Ide L, Parker D. Hazardous child labor: lead and neurocognitive development. Public health reports. 2005;120(6):607–12.
51. Kumar A, Clark CS. Lead loadings in household dust in Delhi. Indoor Air. 2009;19(5):414-20.
52. Warniment C, Tsang K, Galazka SS. Lead poisoning in children. Am Fam Physician. 2010 Mar 15;81(6):751-7.
53. Ravi R. Lead toxicity as a result of herbal medication. Indian Journal of Clinical Biochemistry. 2008;23(2):200-20.
54. Al-Saleh. Determinants of blood lead levels in Saudi Arabian schoolgirls. Int J Occup Environ Health. 1999;5(2):107-14.
55. Mattel Re- calls Various Barbie R Accessory Toys Due to Violation of Lead Paint Standard: U.S. Consumer Product Safety Commission news. ; 2007 [cited 2008 September 4]. Available from: http://www.cpsc.gov/cpscpub/prerel/prhtml07/07301.html.
56. Death of a child after ingestion of a metallic charm Minnesota: CDC 2006. 340-1
57. China exports lead poisoning 2013 [Dec 23]. Available from: http://www.wnd.com/2007/06/41959/.
58. Montgomery M, Mathee A. A preliminary study of residential paint lead concentrations in Johannesburg. Environ Res J. 2005;93(3):279– 83.
59. World Health Organization. Brief guide to analytical methods for measuring lead in paint. Geneva: WHO; 2011. 8 p.
60. Pediatric environmental health. 2 ed. Illinois: American Academy of Pediatrics; 2003.
61. Mahaffey K. Nutrition and lead: strategies for public health. Environ Health Perspect. 1995;103:191-6.
62. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. Environ Res. 1994;65:42-55.
63. Toxicological Profile for lead. Atlanta: Agency for Toxic Substances and Disease Registry-US Department of Health and Human Services; 1999.
64. Pediatric environmental health. 2 ed. Elk Grove Village, IL: American Academy of Pediatrics Committee on Environmental Health -American Academy of Pediatrics.; 2003.

65. Wu T, Buck G, Mendola P. Blood lead levels and sexual maturation in U.S. girls: the Third National Health and Nutrition Examination Survey, 1988–1994. . Environmental health perspectives. 2003;111:737–41.
66. Pilsner J. Influence of prenatal lead exposure on genomic methylation of cord blood DNA. Environmental health perspectives. 2009;117:1466–71.
67. Basha M. The fetal basis of amyloidogenesis: exposure to lead and latent overexpression of amyloid precursor protein and ß-amyloid in the aging brain. J Neurosci. 2005 (25):823–9.
68. Schneider J, DeCamp E. Postnatal lead poisoning impairs behavioral recovery following brain damage. Neurotoxicology, 2007;28(6):1153–1157.
69. Needleman HL. Lead poisoning in children: neurologic implications of widespread subclinical intoxication. Seminars in psychiatry. 1973 Feb;5(1):47-54.
70. Rogan W. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. N Engl J Med. 2001;344(19):1421–6.
71. Byers R, Lord E. Late effects of lead poisoning on mental development. Am J Dis Child. 1943;66:471–94.
72. Schwaetz J, Landrigan P, Baker E. Lead induced anemia: dose response relation and evidence for a threshold. Am J Public Health. 1990;80:165-8.
73. Turgut S, Polat A, Inan M, Turgut G, Emmungil G, Bican M. Interaction between anemia and blood levels of iron, zinc, copper, cadmium and lead in children. Indian J Pediatr. 2007;74:827-30.
74. Needleman H, Schell A, Bellinger D, Leviton A, Allred E. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. The New England journal of medicine. 1990;322:83-8.
75. Needleman H, Riess J, Tobin M, Biesecker G, Greenhouse J. Bone lead levels and delinquent behavior. JAMA. 1996;275:363-9.
76. Sciarill W, Alexander G, Farrell K. Lead exposure and child behavior. Am J Public Health. 1992;82:1356-60.
77. Pocock S, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. BMJ. 1994;309:1189-97.
78. Koller K, Brown T, Spurgeon A, Levy L. Recent developments in low-level lead exposure and intellectual impairment in children. Environ Health Perspec. 2004;112:987–94.
79. Palaniappan K, Roy A, Balakrishnan K, Krishnan L, Mukherjee B, Huand H, et al. Lead Exposure and Visual-Motor Abilities in Children from Chennai, India. Neurotoxicology. 2011 August;32(4):465–70.
80. Canfield R. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. N Engl J Med. 2003;384(16):1517–26.
81. Lanphear B. Cognitive deficits associated with blood lead concentrations <10 mg/dL in US children and adolescents. 2000;Public Health Reports(115):521–9.
82. Bellinger D, Needleman H. Intellectual impairment and blood lead levels. N Engl J Med. 2003;349:500.
83. Needleman H. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Engl J Med. 1979;300(13):689–95.
84. Colborn T, Dumanoski D, Myers J. Our stolen future: are we threatening our fertility, intelligence, and survival? – a scientific detective story. London: Little Brown and Company; 1996.
85. Nevin R. How lead exposure relates to temporal changes in IQ, violent crime and unwed pregnancy. Environ Res J. 2000;89:1-22.

86. Pirkle J. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. Am J Epidemiol. 1985;121:246–58.
87. Satin K, Neutra R, Guirguis G, Flessel P. Umbilicial cord blood levels in California. . Arch Environ Occup Health. 1991;46:167–73.
88. Ghai O, Gupta P, Paul V. Ghai essential paediatrics. 5 ed. New Delhi2003.
89. Goyer R. Results of lead research: prenatal exposure and neurological consequences. Environ Health Perspect. 1996;104:10-50.
90. Kumar BR, Dudal SR, Rao A. Kuppuswamy's socio-economic status scale – a revision of economic parameter for 2012. Int J Resear Dev Health. 2013;1(1):2-4.
91. Feng T, Keller LR, Wang L, Wang Y. Product Quality Risk Perceptions and Decisions: Contaminated Pet Food and Lead-Painted Toys. Risk Analysis 2010; 30(10):1572-1589.
92. Kumar A, Pastore P. Lead and cadmium in soft plastic toys. Current Science 2007; 93(6):818-822.

13 Annexures

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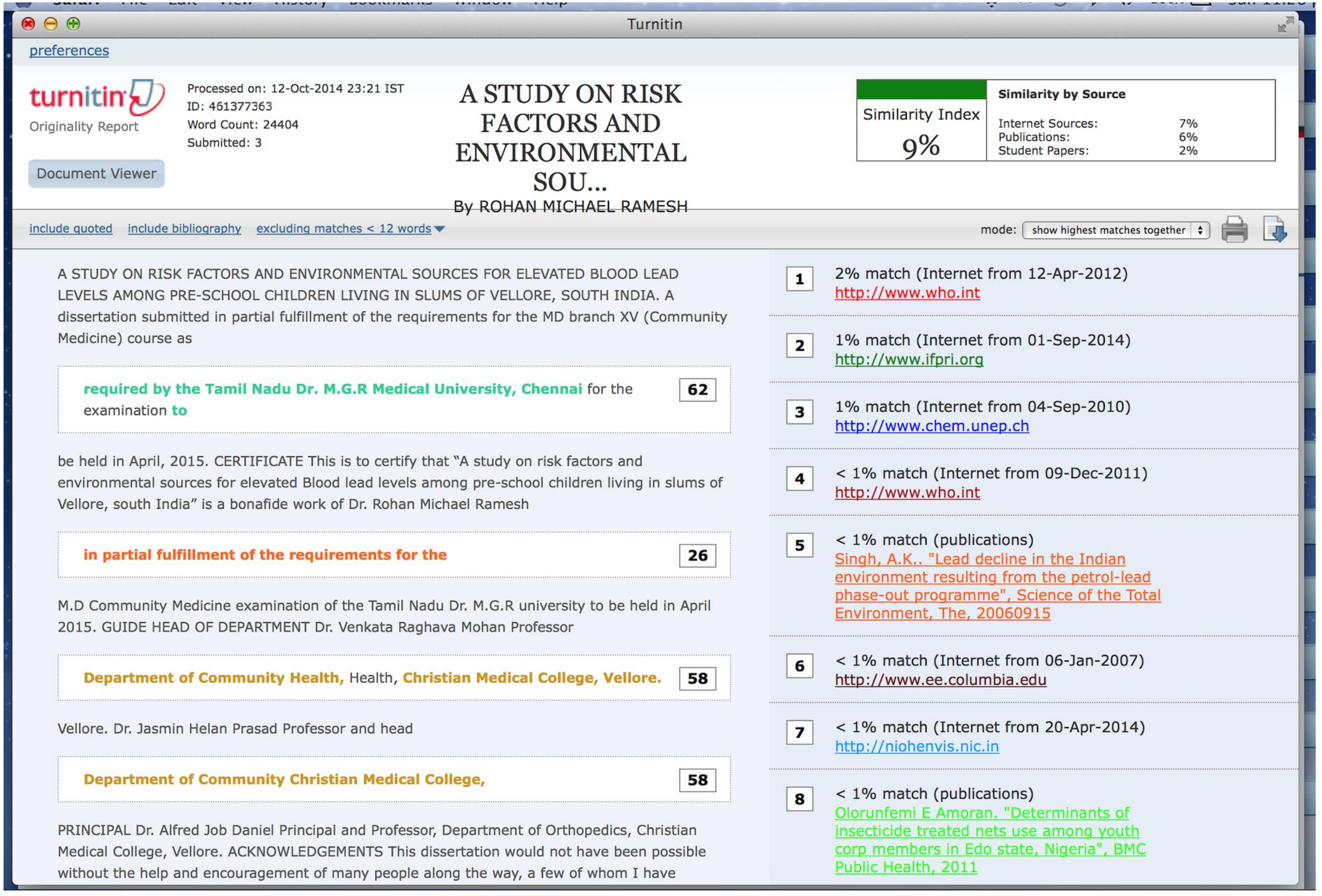

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A STUDY ON RISK FACTORS AND ENVIRONMENTAL SOURCES FOR ELEVATED BLOOD LEAD LEVELS AMONG PRE-SCHOOL CHILDREN LIVING IN SLUMS OF VELLORE, SOUTH INDIA. A dissertation submitted in partial fulfillment of the requirements for the MD branch XV (Community Medicine) course as

required by the Tamil Nadu Dr. M.G.R Medical University, Chennai for the examination to — 62

be held in April, 2015. CERTIFICATE This is to certify that "A study on risk factors and environmental sources for elevated Blood lead levels among pre-school children living in slums of Vellore, south India" is a bonafide work of Dr. Rohan Michael Ramesh

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PRINCIPAL Dr. Alfred Job Daniel Principal and Professor, Department of Orthopedics, Christian Medical College, Vellore. ACKNOWLEDGEMENTS This dissertation would not have been possible without the help and encouragement of many people along the way, a few of whom I have

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8. < 1% match (publications) Olorunfemi E Amoran. "Determinants of insecticide treated nets use among youth corp members in Edo state, Nigeria", BMC Public Health, 2011

Annexure 2:Questionnare

QUESTIONNAIRE

A study on 'Risk factors and environmental causes of elevated blood lead levels in pre-school children'

Section A: Demographic details

Study Id no: Phone number:
Area name:
Name of the child:
Informant's name and relationship:
DOB:
Sex:
Address:
Caste:
Religion:
Type of house:
Type of roof……………./ wall…………../floor…………………
Number of rooms:
Total number of people

Sl no.	Name	Relationship to participant	Age (Yrs)	Sex	Occupation	Yrs. Of Occupation	Income per Month	Education

Section B: Familial exposure

High risk occupations:

Plumbers, pipe fitters
Lead miners
Lead smelters and refiners
Auto repairers
Glass manufacturers
Shipbuilders
Printers
Plastic manufacturers
Police officers
Steel welders or cutters
Construction workers (especially renovation and rehabilitation)
Rubber product manufacturers
Gas station attendants (past exposure)

Battery manufacturers
Battery recyclers
Bridge reconstruction workers
Firing range instructors
Painter
Electrician/electronics

Sl. no	Name	Occupation	Durati on of	Paint Yes/	Tyre Yes/	Battery Yes/ no	Work Yes/ no	Change clothes after work	Bath /day	Bath Before/ After work
1										
2										
3										
4										

Section C: House details

1. Paint details:

Sl no.	Area	No. of rooms	Painted	White washed	Both	Last painted	How many times in the last 5 years	Brand of the paint	Colour	Quantity
1	House									
2	Roof									
3	Floor									
4	Walls									
5	Windows									
6	Doors									
7	Others…..									

1. State of the house: Age of house:
    i. New (1year)
    ii. Old
    iii. Recently renovated (within one year)
    iv. Under construction/renovation:
2. Is there a battery shop / pottery unit / factory / battery near your house?
    a. How far is it from your house?
    b. Does your child play near that shop?
    c. Does your child play with tyres/pottery/battery
    ***Observation by the investigator regarding the area:
3. How often do you sweep the house?
    a. Twice a day
    b. Once a day
    c. Alternate days
    d. ____times a day

4. What do you use to clean the house with?
    a. Water only
    b. Water and soap
    c. Water and _______
    d. Sweep only

   How often?

Section D: Habits

1. Does your child have a habit of licking, chewing or eating various objects in the house?
    a. List the objects:
    b. Has the object been recently painted? (Yes/No)
    c. Is the paint peeling from the object? (Yes/No)
    d. Color of the paint used in that object?
    e. Does your child lick or eat mud/dirt? (Yes/No)
        i. From where? (Indoor/Outdoor)
    f. Does your child chew windowsills and door edges? (Yes/No)
    g. Does your child chew pencils/crayons? (Yes/No)
    h. Does your child chew chocolate wrappers? (Yes/No)
    i. Nail biting : (Yes / No)
    j. Pepsicola ice (popsicle): (yes/ no)
        i. How many:

       **If available for observation at the house, then investigator has to mention

2. Do you apply kajal/ surma / eye cosmetics /sindoor / lipstick for your child?
    a. Color:
    b. How many times per day:
    c. How many days a week:
    d. Do you wash it off at the end of the day? (Yes/No)
        i. If yes, how?
    e. Brand of the product:

3. Is your child still breastfeeding? (Yes/ no)
    1. EBF how long?

4. Do you give your child Ayurvedic/alternative /native medications?
    a. How often:
        i. Once a week
        ii. Once a month
        iii. Rarely
        iv. Never
    b. For which conditions:
    c. Where do u buy these medications:

5. Toys/article Details:

Sl no.	Risk articles	Observer Details	Material	Duration (Mins)	Regularity per day	Behaviour (eating/biting/licking)
1						
2						
3						
4						
5						
6						

6. Daily activities of the child
   a. Can you list out all the activities the child does routinely every day:
      i. Morning:
      ii. Afternoon:
      iii. Evening:
      iv. Night:
   b. Can you mention 3 activities that your child your child predominantly does every day
      i. :
      ii. :
      iii. :
   c. Do you wash your child's hands before eating? (YES / NO)
   d. Do you wash your child's hands with soap before eating? (YES / NO)
   e. Hand to mouth activities of the child: (YES / NO)
   f. Eating from vendors: (YES / NO)

Section E- Ingestion

Water source:

(a) Source of drinking water :
   i. Municipal
   ii. Bore well
   iii. Open well
   iv. Payed tankers
   v. Others:

1) In what vessels do you store drinking water in?copper /iron /plastic/ Steel/ Aluminum/brass

2) Do you boil it before the children drink? (YES / NO)

3) What type of **tap** do you get the source of water from? Brass/ lead/ plastics/**Iron/NA**

4) What type of **pipe** do you get the source of water from? Brass/ lead/ plastics/**Iron/open pipe**

5) Food habit: veg / non veg

6) Frequency of meals: once /twice /thrice a day/____________a day

7) Do you use ceramic containers coated with enamel in your house? (YES / NO)

   a) What do you use it for?

   b) How often do you use it?

8) Do you use melamine plates/dishes/cups coated with enamel in your house?

   (YES / NO)

   a) What do you use it for?

   b) How often do you use it?

9) Do you use aluminum containers in your house? (YES / NO)

   a) What do you use it for?

   b) How often do you use it?

10) Do you use stainless steel containers in your house? (YES / NO)

    a) What do you use it for?

    b) How often do you use it?

11) Do you use tinned brass containers in your house? (YES / NO)

    a) What do you use it for?

    b) How often do you use it?

Section F- Misc. .

1. Smokers in the family: Y/N (How many……)
   a. Smoking inside the house: (YES / NO)
   b. Cigarette / Beedi
   c. How many per day:
   d. Duration in years:

Section G-Sample details

sampleID	type	Details	Date of collection	quantity

Annexure 3: Information sheet (Tamil)

தகவல் படிவம்

பள்ளிவயதை எட்டாத சிறுவர்களின் இரத்தத்தில் அதிக ஈயம் இருப்பதற்கு காரணமான சூழ்நிலை பற்றிய ஆராய்ச்சி :

தகவல் படிவம் : இந்த ஆராய்ச்சியைப்பற்றியும் இதில் உங்கள் பங்கேற்றலைப் பற்றியும் கீழ்க்காணும் தகவல்கள் உங்களுக்கு அளிக்கப்படுகிறது. இதை கவனமாக படித்து இதைப்பற்றிய கேள்விகளை நீங்கள் தயங்காமல் கேட்கலாம் இந்த தகவல் படிவம் உங்களுக்கு அளிக்கப்பட்டு இதைப்பற்றிய கேள்விகளை நீங்கள் கேட்கும் தருணம் அளிக்கப்பட்டு, அவற்றிற்கான பதில்கள் உங்களுக்கு தரப்படும். இந்த ஆராய்ச்சியில் உங்கள் பங்கெடுப்பு முற்றிலும் உங்கள் சுயவிருப்பமே. நீங்கள் இதிலிருந்து எப்போது வேண்டுமானாலும் தாராளமாக விலகிக் கொள்ளலாம். அப்படி நீங்கள் விலகினாலும் அது இந்த மருத்துவமனையில் நீங்கள் பெறும் எந்த சிகிச்சையையும் பாதிக்காது. (சி.எம்.சி, வேலூர்/ LCECU/ CHAD மருத்துவமனை)

ஆராய்ச்சியின் நோக்கம் : MalEd என்ற ஆராய்ச்சியில் சில குழந்தைகளுக்கு இரத்தத்தில் ஈயத்தின் அளவு, சரியான அளவைவிட கூடுதலாக இருப்பது கண்டறியப்பட்டது. இதன் காரணமாக மருத்துவ மற்றும் மனநிலை ரீதியாக சில பாதிப்புகள் ஏற்படலாம். ஆகையால், இது கவனம் செலுத்தப்படவேண்டிய ஒரு விஷயம்.

இந்த ஆராய்ச்சியின் மூலம் உங்கள் குழந்தையின் உடலில் ஈயம் அளவு அதிகமானால் ஏற்படக்கூடிய பாதிப்புகளுக்கு காரணமாக இருக்கும் கூறுகளை கண்டறிந்து அதை தடுப்பது இதன் நோக்கம்.

கடைப்பிடிக்க வேண்டிய முறைகள்:- உங்கள் குழந்தை MalEd ஆராய்ச்சியில் பங்கேற்று இருப்பதால் உங்கள் குழந்தையின் பிறப்பு பற்றிய விவரங்கள், உணவு மற்றும் பழக்கங்கள் உடல்நலம், தடுப்பு ஊசி விவரம் மற்றும் சில அன்றாட விவரங்கள் குறித்து நாங்கள் கேள்விகள் கேட்போம். மேலும், உங்கள் குடும்ப உறுப்பினர்கள் பற்றியும் உங்களது பொருளாதார நிலை குறித்தும் நாங்கள் உங்களை வினவுவோம். உங்களை வீடுகளில் வந்து சந்திக்கும் மருத்துவர் இந்த கேள்விகளை கேட்பார்.

ஆராய்ச்சியின் கால வரையறை: ஓர் ஆண்டு (டிசம்பர் 2013 முதல் நவம்பர் 2014 வரை)

எதிர்பார்க்கும் செலவு : உங்களுக்கு செலவு எதுவும் கிடையாது.

இதில் பங்கேற்பதினால் உங்களுக்கு ஏற்படக்கூடிய அசௌகரியங்கள் / விபரீத விளைவுகள் பற்றிய விவரம் :
எங்களுடைய சில கேள்விகள் உங்களது குழந்தை மற்றும் உங்களைப் பற்றிய தனிப்பட்ட வாழ்வுக்குரியதாக இருப்பதால், ஒரு வேளை நீங்கள் அசௌகரியமாக உணரலாம்.

எதிர்பாராத அபாயங்கள் : அப்படி எந்த ஒரு எதிர்பாராத அபாயமும் உங்களுக்கோ உங்கள் குழந்தைக்கோ ஏற்படாது.

ஆராய்ச்சியின் போது பாதிப்பு ஏற்பட்டால் அதற்கான நஷ்ட ஈடு :
இந்த ஆராய்ச்சியில் அப்படி பாதிப்பு ஏற்பட வாய்ப்பு இல்லை என நாங்கள் நம்புவதால், நஷ்ட ஈடு எதுவும் தரப்படமாட்டாது.

ஆராய்ச்சியில் பங்குபெற்றால் எதிர்பார்க்கப்படும் நன்மைகள்:
குழந்தையின் உடலில் ஈயத்தினால் ஏற்படக்கூடிய விஷத்தன்மைக்கு காரணமான கூறுகளை கண்டறிந்தால், பலவிதமான உடல்நல குறைவுகளை உங்கள் குழந்தை எதிர்கொள்ளாமல் தடுக்க இயலும். உங்கள் குழந்தை மட்டுமல்ல, இதே சூழ்நிலையில் வாழும் மற்ற குழந்தைகளும் இதன் மூலம் நன்மை அடைவார்கள்.

மாற்று சிகிச்சை முறை: கிடையாது

இதில் பங்கு பெற்றால் தரப்படும் ஈட்டு தொகை: நாங்கள் கேட்கும் கேள்விகளுக்கு நீங்கள் பதில் சொல்வதற்கோ, இந்த ஆராய்ச்சியில் நீங்கள் பங்கெடுப்பதற்கோ பணம் தரப்படமாட்டாது.

ஆராய்ச்சியின் தலைவர் இந்த ஆராய்ச்சியிலிருந்து உங்களை நீக்கிவிடக்கூடிய சூழ்நிலைகள்: நாங்கள் கேட்கும் கேள்விகளுக்கு பதில் கூற உங்களுக்கு விருப்பம் இல்லாவிடிலோ அல்லது நாங்கள் உங்கள் இல்லத்திற்கு வருவது உங்களுக்கு பிடிக்கவில்லை என்றாலோ இதிலிருந்து நீங்கள் விலகிக் கொள்ளலாம்.

நீங்கள் விலகிக் கொண்டால் என்ன நேரிடும் : எங்களுக்கு நீங்கள் அளித்த தகவல்களை நாங்கள் பயன்படுத்தாமல் அவற்றை அழித்து விடுவோம்.

தகவல் தொடர்பு :
இதைப்பற்றிய உங்கள் கேள்விகளை தயங்காமல் கீழ்க்காணும் நபர்களிடம் கேட்கலாம்.
டாக்டர். **ரோஹன் ரமேஷ் + 91 9600690072,**
டாக்டர். **வெங்கட்ராகவ் +919443109044**

நம்பகம் : உங்களது தகவல்கள் அனைத்தும் இந்த ஆராய்ச்சியில் இரகசியமாக காக்கப்பட எல்லா முயற்சிகளும் எடுக்கப்படும்.

இரகசிய காப்பு : உங்களது தகவல்கள் அரசு மற்றும் கிறிஸ்தவ மருத்துவ மனையின் நிர்வாகக் குழுவிடம் பகிர்ந்து கொள்ளப்படலாம்.

Annexure 4: Consent (Tamil)

ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு :

6 முதல் 36 மாத வயது வரம்புக்குட்பட்ட குழந்தைகளின் இரத்தத்தில் ஈயத்தின் அளவு அதிகரிப்புக்கான காரணங்கள்.

தேதி :

தென் இந்திய சமுதாய அடிப்படையிலான
ஆராய்ச்சி பங்கு பெறுபவர் பெயர் : - தாய் : ....

கணவர் பெயர் : .... வயது : ....

கிராமம்/நகர்ம : ....

இந்த ஆராய்ச்சியை செய்பவர் மூலம், சிறு குழந்தைகளின் இரத்தத்தில் ஈயத்தின் அளவு அதிகரித்தால் ஏற்படக்கூடிய அபாயங்கள் பற்றி எனக்கு தகவல்கள் கூறப்பட்டது. அவ்வாறு ஈயத்தின் அளவு இரத்தத்தில் அதிகரித்தால் உயிருக்கு ஆபத்து ஏற்படும். இந்த ஆராய்ச்சியின் விளைவினால் அத்தகைய இழப்பு ஏற்படாமல், திட்டங்கள் வகுக்க இயலும், என்பதை விளங்கி கொண்டேன்.

என் குழந்தையை பற்றிய விவரங்கள், குறித்த கேள்விகள் அந்த நாளில்தானே கேட்கப்பட்டு முடியும் என்பதை அறிந்துக் கொண்டேன். சில கேள்விகள் எனக்கு ஒருவித அசௌகரியத்தை ஏற்படுத்தி மன உளைச்சலை ஏற்படுத்தலாம் என்றும் விளங்கிக் கொண்டேன்.

இதையன்றி எனக்கோ / என் குடும்ப உறுப்பினர்களுக்கோ / என் குழந்தைக்கோ, வேறு எந்த பாதிப்பும் ஏற்படாது என அறிகிறேன். நான் அளித்துள்ள தகவல்கள் அனைத்தும் இரகசியமாக காக்கப்பட்டு ஆராய்ச்சிக்காக மட்டுமே பயன்படுத்தப்படும் என அறிகிறேன். எனினும் ஆராய்ச்சியின் முடிவுகள் அரசாங்க அதிகாரிகளிடம் பகிர்ந்துக் கொள்ளப்படலாம். இந்த அராய்ச்சியில் எனது பங்கேற்பு முற்றிலும் எனது சுயவிருப்பம் என அறிகிறேன். இதில் தொடர்ந்து பங்குபெற நான் விரும்பாமல் விலகிக் கொண்டால் இந்த ஆராய்ச்சி செய்வரின் நிறுவனத்தில் எனக்கு தற்போது அளிக்கப்படும் பராமரிப்பு எதிர்காலத்திலும் எந்த விதத்திலும் பாதிக்கப்படாது. (சி.எம்.சி, வேலூர் LCECU / CHAD மருத்துவமனை) இந்த ஆராய்ச்சியில் நான் பங்கெடுக்கும் சமயத்தில் மருத்துவ அல்லது மன ரீதியாக நான் பாதிக்கப்பட்டால் என் விருப்பத்திற்கு இணங்க எனது பொருளாதார நிலைக்கு ஏற்ப LCECU (அ) CHAD மருத்துவமனையில் ஆராய்ச்சியாளர் எனது சிகிச்சைக்கான ஏற்பாடுகளை செய்வார் என்றும் கூறப்பட்டது.

அடைப்புகளை பூர்த்தி செய்க :

1. நான் இந்த தகவல் படிவத்தை படித்து, புரிந்துக் கொண்டு, கேள்விகளை கேட்க தருணம் அளிக்கப்பட்டேன் என உறுதி செய்கிறேன் ( )

2. இந்த ஆராய்ச்சியில் எனது பங்கேற்பு முற்றிலும் என் சுயவிருப்பம் என்றும், நான் எப்போது வேண்டுமானாலும் எந்த காரணமும் கூறமல் இதிலிருந்து விலகிக் கொண்டாலும் அது எனது மருத்துவ உரிமைகளை பாதிக்காது என்றும் விளங்கிக் கொண்டேன் ( )

3. இந்த ஆராய்ச்சியில் என்னை பற்றின தகவல்களை எனது அனுமதி இன்றி ஆராய்ச்சியாளர்கள் நோக்கலாம் என்றும் மேலும் ஏதாவது ஆராய்ச்சிகளுக்கு அவற்றை பயன்படுத்திக் கொள்ளலாம் என்றும் அறிகிறேன், அதற்கு சம்மதிக்கிறேன் ஆனாலும் எனது அடையாளம் வெளியிடபடமாட்டாது என்று அறிகிறேன். ( )

4. விஞ்ஞான சம்பந்தப்பட்ட தேவைகளுக்கு இந்த ஆராய்ச்சியின் முடிவுகளை உபயோகப்படுத்துவதற்கு நான் தடை செய்ய மாட்டேன் ( )

5. மேற்கூறிய ஆராய்ச்சியில் பங்கு பெற நான் ஒப்புதல் அளிக்கிறேன் ( )

இந்த ஆராய்ச்சியைப் பற்றிய கேள்விகள் ஏதாவது இருப்பின் கீழ்க்காணும் நபர்கள் தொடர்பு கொள்ளவும்
டாக்டர். **ரொகன் ரமேஷ் + 91 9600690072,** டாக்டர். **வெங்கட்ராகவன் +919443109044**

உங்களின் பங்கேற்றல் உங்கள் சுயவிருப்பமே நீங்கள் பங்கு பெறவிட்டாலும், விலகிக் கொள்ள தீர்மானித்தாலும் நீங்கள் தண்டிக்கப்படவோ அல்லது உங்களது சலுகைகள் மறுக்கபடவோ மாட்டாது. இந்த படிவத்தில் நீங்கள் கையொப்பம் இடுதல் இந்த ஆராய்ச்சியைப்பற்றி உங்களுக்கு தகவல்கள் விளக்கமாக கூறப்பட்டு, நீங்கள் மனமுவந்து இதில் பங்கேற்க ஒப்புதல் அளிப்பதற்கு அர்த்தமாகும்.

பங்கேற்பவரின் கையொப்பம்	கட்டைவிரல் அடையாளம்
பங்கேற்பவரின் பெயர் :	தேதி
சாட்சியின் பெயர் :	சாட்சிகள் கையொப்பம்
	தேதி :
ஆராய்ச்சி நடத்துபவரின் கையொப்பம்,	தேதி :

Annexure 5:Vellore institute of technology-Permission letter

R- 4250

To
The Pro-VC
VIT University
Vellore

Dr. P. K. 30/12

Subject:-To grant permission to collaborate with CMC Vellore
Ref:- Request from Dr Venkata Raghava M. MD, MPH, (CMC Vellore) to accept as Co-Invetigator in his Research Project along with his PG Student Dr Rohan

Respected Sir,

I would like to bring your kind notice that, Dr Venkata Raghava M. MD, MPH, **(CMC Vellore), has requested me to** accept as a Co-Invetigator in his Research Project along with his PG Student Dr Rohan. This group is working on **"A study of the risk factors and environmental sources for elevated blood lead levels among children who have completed 24 months of age in an urban slum in South India."**.

In order to involve my name, **Institutional Review Board, Christian Medical College, Vellore, needs Official approval from VIT University Vellore.**

I would like to highlight that this will help to generate some research papers, along with further strengthening of bond of VIT-CMC.

Kindly grant permission to join the team for collaboration. The outcome of this will be getting bulk samples for lead analysis to TBI. We shall charge to them with some concession as per VIT-TBI rule.

Thanking you.

Dr Pundlik Rambhau Bhagat
9047289073

Can we permit them sir?

Submitted to VC for approval pl

30/12

To Registrar, ProVC, & Dean SAS.
- OK to partner with CMC.
- Please be careful to follow all CMC norms and procedures.
- The VIT-CMC partnership is very important to us. So anything we do in dealing with samples and other items, we should follow proper procedures.
- Since the request has come from CMC, it is even more important to do everything right.

Dec. 30/13

Enclosures:-

[i] Requisition from Dr Venkata Raghava M. MD, MPH, (CMC Vellore)
[ii] Research proposal prepared by Dr Rohan [3 pages]
[iii] Address of Dr Venkata Raghava M. MD, MPH, with cell for your convenience

Dean, SAS
30/12

Annexure 6: IRB Clearance Certificate

OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE, VELLORE, INDIA.

Dr. B.J. Prashantham, M.A., M.A., Dr. Min (Clinical)
Director, Christian Counseling Center,
Chairperson, Ethics Committee.

Dr. Alfred Job Daniel, D Ortho, MS Ortho, DNB Ortho
Chairperson, Research Committee & Principal

Dr. Nihal Thomas,
MD., MNAMS., DNB (Endo), FRACP (Endo), FRCP (Edin), FRCP (Glasg)
Deputy Chairperson
Secretary, Ethics Committee, IRB
Additional Vice Principal (Research)

April 14, 2014

Dr. Rohan Michael Ramesh
PG Registrar
Department of Community Health
Christian Medical College
Vellore 632 004

Sub: **Fluid Research grant project:**
A study of risk factors and environmental sources for elevated blood lead levels among pre-school children living in slums of Vellore, southern India. Dr. Rohan Michael Ramesh, PG Registrar, Community Health, Dr. Venkata Raghava, Community Health, Dr. Anuradha Rose, Community Health, Dr. Gagandeep Kang, Gastrointestinal Sciences, Dr. Sushil Mathew John, Low Cost Effective Care Unit, CMC, Vellore / Dr. Pundlik R Bhagat, Organic Chemistry Division, Program Chair, M Sc. Chemistry, School of Advanced Sciences, V.I.T University, Vellore.

Ref: IRB Min No: 8610 [OBSERVE] dated 07.01.2014

Dear Dr. Rohan Michael Ramesh,

I enclose the following documents:-

1. Institutional Review Board approval 2. Agreement

Could you please sign the agreement and send it to Dr. Nihal Thomas, Addl. Vice Principal (Research), so that the grant money can be released.

With best wishes,

Dr. Nihal Thomas
Secretary (Ethics Committee)
Institutional Review Board

Dr. NIHAL THOMAS
MD.,MNAMS.,DNB(Endo),FRACP(Endo),FRCP(Edin),FRCP(Glasg)
SECRETARY - (ETHICS COMMITTEE)
Institutional Review Board,
Christian Medical College, Vellore - 632 002

1 of 5

Cc: Dr. Venkata Raghava, Community Health, CMC, Vellore

Christian Medical College, Vellore, Tamil Nadu 632 002.

OFFICE OF RESEARCH
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Ref: IRB Min No: 8610 [OBSERVE] dated 07.01.2014

Dear Dr. Rohan Michael Ramesh,

The Institutional Review Board (Blue, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project entitled "A study of risk factors and environmental sources for elevated blood lead levels among pre-school children living in slums of Vellore, southern India." on January 7th 2014.

The Committees reviewed the following documents:

1. IRB Application format
2. Curriculum Vitae' of Drs. Rohan Michael Ramesh, Venkata Raghava, Anuradha Rose, Gagandeep Kang, Sushil Mathew John, Pundlik R Bhagat.
3. Information sheet (English & Tami)
4. Consent form (English & Tamil)
5. Questionnaire (English & Tamil)
6. No of documents 1-5

2 of 5

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Deputy Chairperson
Secretary, Ethics Committee, IRB
Additional Vice Principal (Research)

The following Institutional Review Board (Blue, Research & Ethics Committee) members were present at the meeting held on January 7th 2014in the CREST/SACN Conference Room, Christian Medical College, Bagayam, Vellore 632002.

Name	Qualification	Designation	Other Affiliations
Dr. T. Balamugesh	MBBS, MD(Int Med), DM, FCCP (USA)	Professor, Pulmonary Medicine, CMC, Vellore	Internal, Clinician
Dr. Mathew Joseph	MBBS, MCH	Professor, Neurosurgery, CMC, Vellore	Internal, Clinician
Dr. J. Visalakshi	MPH, PhD	Lecturer, Dept. of Biostatistics, CMC, Vellore	Internal, Statistician
Dr. Susanne Abraham	MBBS, MD	Professor, Dermatology, Venerlogy & Leprosy, CMC, Vellore	Internal, Clinician
Dr. Ranjith K Moorthy	MBBS M Ch	Professor, Neurological Sciences, CMC, Vellore	Internal, Clinician
Dr. Vivek Mathew	MD (Gen. Med.) D.M (Neuro) Dip. NB (Neuro)	Professor, Neurology, CMC, Vellore	Internal, Clinician
Mrs. Shirley David	M.Sc, PhD	Professor, Head of Fundamentals Nursing Department, CMC, Vellore	Internal, Nurse
Mrs. Pattabiraman	B. Sc, DSSA	Social Worker, Vellore	External, Lay person
Mr. C. Sampath	B. Sc, BL	Legal Expert, Vellore	External, Legal Expert
Dr. Ebenezer Ellen Benjamin	M.Sc, PhD	Professor, Maternity Nursing, CMC, Vellore	Internal, Nurse

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Deputy Chairperson
Secretary, Ethics Committee, IRB
Additional Vice Principal (Research)

Dr. B. J. Prashantham	MA(Counseling Psychology), MA(Theology), Dr. Min(Clinical Counselling)	Chairperson, Ethics Committee, IRB. Director, Christian Counseling Centre, Vellore	External, Social Scientist
Dr. Jayaprakash Muliyil	B. Sc, MBBS, MD, MPH, Dr PH (Epid), DMHC	Retired Professor, Vellore	External, Scientist & Epidemiologist
Dr. Denise H. Fleming	B. Sc (Hons), PhD	Honorary Professor, Clinical Pharmacology, CMC, Vellore	Internal, Scientist & Pharmacologist
Rev. Joseph Devaraj	B. Sc, BD	Chaplaincy Department, CMC, Vellore	Internal, Social Scientist
Dr. Nihal Thomas,	MD, MNAMS, DNB(Endo), FRACP(Endo) FRCP(Edin) FRCP (Glasg)	Professor & Head, Endocrinology. Additional Vice Principal (Research), Deputy Chairperson, IRB, Member Secretary (Ethics Committee), IRB, CMC, Vellore	Internal, Clinician

We approve the project to be conducted as presented.

The Institutional Ethics Committee expects to be informed about the progress of the project, any **adverse events** occurring in the course of the project, any **amendments in the protocol and the patient information / informed consent**. On completion of the study you are expected to submit a copy of the **final report**. Respective forms can be downloaded from the following link: http://172.16.11.136/Research/IRB_Polices.html in the CMC Intranet and in the CMC website link address: http://www.cmch-vellore.edu/static/research/Index.html.

IRB Min No: 8618 [OBSERVE] dated 07.01.2014

OFFICE OF RESEARCH
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CHRISTIAN MEDICAL COLLEGE, VELLORE, INDIA.

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Deputy Chairperson
Secretary, Ethics Committee, IRB
Additional Vice Principal (Research)

Fluid Grant Allocation:

A sum of Rs. 50,000/- INR (Rupees Fifty Thousand only) will be granted for 1 year.

Yours sincerely

Dr. Nihal Thomas
Secretary (Ethics Committee)
Institutional Review Board

Dr. NIHAL THOMAS
MD.,MNAMS.,DNB(Endo),FRACP(Endo),FRCP(Edin),FRCP(Glasg)
SECRETARY - (ETHICS COMMITTEE)
Institutional Review Board,
Christian Medical College, Vellore - 632 002.

Cc: Dr. Venkata Raghava, Community Health, CMC, Vellore

IRB Min No: 8618 [OBSERVE] dated 07.01.2014

Annexure 7: Photographs